



# CARDIOVASCULAR SOUND

## IN HEALTH AND DISEASE



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## IN HEALTH AND DISEASE

*Being a Comprehensive Treatise Introduced by a  
Historical Survey Illustrated Mainly by Sound  
Spectrograms (Spectral Phonocardiograms) and  
Supplemented by an Extensive Bibliography*

*With a Section on Respiratory Sound*

By

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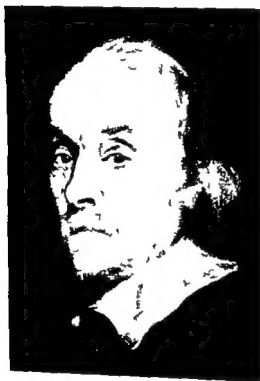
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*Dedicated to the memory of*

**WILLIAM HARVEY**

(1578-1657)

*whose reference to the heart sounds is the  
earliest and clearest which survives  
in the Tercentennial Year after his death*



WILLIAM HARVEY (1578-1657)

Detail Royal College of Physicians portrait (189)



# Preface

Any virtue which this monograph may have will probably be related to one or all of the following features: (1) the comprehensive and I trust critical historical survey; (2) the use of spectral phonocardiogram for purposes of illustration; (3) discussion of auscultatory signs on the basis of physical and physiological principles whenever possible in the present state of knowledge; (4) an obsession with numbers graphed relation ship and quantification in this field which is still largely qualitatively descriptive; and finally (5) the provision of an extensive bibliography.

Cardiovascular sound and cardiac cultivation are at the hub of clinical cardiology and cardiovascular physiology. The findings of so many other diagnostic methods have permanence in connection with cardiovascular sound and vice versa and in a few cardilogic condition is cardiovascular sound of no ignificance that in exhaustive treatise on cardiovascular sound approaches being a textbook of cardiology. For this an apology is offered.

Why cultivate the art of cardiac cultivation when study the science of cardiovascular sound in these days of cardiac catheterization and angiocardiology? In great measure is that cardiovascular sound as detected by cultivation contains information on cardiovascular function obtainable by no other method. Levine (1957) suggested that the usefulness is mainly of three types: (1) To exclude certain diagnoses, (2) to establish certain diagnoses, (3) to provide clues to diagnostic possibilities requiring exploration. Sometimes it is important to exclude a diagnosis as to establish it. Although rare cases of murmurs, bacterial endocarditis may occur cultivation usually make it possible to exclude this diagnosis with relative certainty

in case of fever of obscure cause. Although there are simulating condition which may deceive the untrained and unchooled ear the diagnosis of pericarditis, aortic regurgitation and mitral stenosis can often be made only by the characteristic auscultatory change. An indication may provide tip to diagnoses which can be established by other method e.g. gallop may suggest that epigastria pain is of cardiac origin.

Spectral phonocardiography appears to have reaching a well scientific value. In the spectral phonocardiogram the display of the frequency spectrum (its unique feature) is responsible for three advantages of the method: (1) Quality, or timbre is given physical definition, (2) resolution in the time dimension is improved, (3) more accurate display of the wide dynamic (intensity) range of cardiovascular sound is obtained. Potentially the method can do all the ear can and probably can even surpass the ear because (1) it is not wed to a particular frequency, (2) it suffers from no psychoacoustic impediments, (3) it provides better resolution in the time dimension and (4) it produce permanent quantifiable record.

The order of presentation that has been adopted is evident from the Table of Contents. A progression from the general to the specific has been practiced. General and basic consideration are first dealt with then the individual elements of cardiovascular sound—transients and murmurs of various types—have been described finally a long synthesizing section discuss the sound phenomena which accompany various categories of cardiovascular disease—valvular, congenital, hypertensive myocardial, pericardial and miscellaneous. The index has been prepared with particular care.

Through a happy coincidence, the year 1957, which was spent in the preparation of this monograph, was also the tercentenary of the death of William Harvey, whose early contribution to the field of cardiovascular sound is described on page 3. It is a pleasure, in dedicating to Harvey this survey of the development of the field in the

last 300 years, to make a small private addition to the various public observances with which cardiologists and cardiovascular physiologists have taken note of the year.

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## SECTION I

### *Historical Survey*



## CHAPTER I

# The History of Cardiovascular Sound<sup>1</sup>

### HARVEY TO LAENNEC

It is surprising that no definite reference to the heart sounds dates from the period before William Harvey. The Hippocratic writings (687a f) of about 400 B.C. indicate that the ear was applied to the chest as an established practice in ancient medicine and was useful in demonstrating what is now referred to as the Hippocratic ecchymosis pla of hydropneumothorax. Other types of medical sound were described: rales (687g h) pleural friction rub (a creak like new leather) (687i j) rhthmic wheezing (212) brachygn (687). Although they must have been heard the heart sound were never commented on.

One might think the sound produced by the normally beating heart was such a common place personal experience that there was contempt for detailed description and analysis. However the derision which greeted description of the heart sound by Harvey and of the fetal heart sound by Marac (p. 4) suggests that existence of the heart sounds was not as common knowledge as one might presume.

It would be supposed that at least abnormal varieties of cardiovascular sound particularly loud murmur audible at a distance from the patient would have excited wonder if they did not record surprise. One can only speculate on how often in primitive and even relatively sophisticated societies the loud raucous murmur of calcific aortic stenosis or of retroverted aortic cup was interpreted as the cries of an evil spirit inhabiting the patient and causing his symptoms.

In 1616 the year that Shakespeare and Cervantes died William Harvey (1578-1657) delivered his visceral lectures. According to his

note which, written in his own hand were discovered in the British Museum Harvey compared the heart to two clicks of a water bellows to raise water (631). He was probably referring to the two sets of heart valves since *clac* was a word used for *take* in Harvey's time. For example the Oxford Dictionary quotes Bate as writing in 1634 "a clicke is a piece of leather navled over my hole having a piece of lead to make it lie close so that the ayre or water in any vessel may thereby bee kept from going out." Harvey's use of an onomatopoeic term *clac* may suggest that he then had knowledge of the heart sound as well as a suspicion of their valvular origin.

In his famous *De Motu Cordis* (1628) Harvey wrote (630) as follows: "with each movement of the heart when there is the delivery of a quantity of blood from the veins to the arteries a pulse takes place and can be heard within the chest."

There can be no doubt that Harvey was referring to an acoustic phenomenon for a contemporary, Aemilius Pirraanus, writing in 1647 stated sarcastically (1180) "Nor we poor doctors nor any other doctor in Venice can hear them, but happy is he who can hear them in London. Duremberg (1816-1872) a competent French medical historian termed this statement a shameful testimony of stupidity (230).

Another clear pre-Laennec reference to the heart sounds is that of Robert Hooke (1633-1703), versatile scientist secretary of the Royal Society of London originator of Hooke's law of elasticity.

I have been able to hear very plainly the beating of a Man's Heart. Who knows I say but that it may be possible to discover the Motions of the Internal

<sup>1</sup> Several general sources (6 a & 1309b 156 156b) were used in the preparation of this section.

Parts of Bodies by the sound they make, that one may discover the Works performed in the several Offices and Shops of a Man's Body, and thereby discover what Instrument or Engine is out of order (710)

In this statement Hooke left no doubt of his familiarity with the heart sounds and in addition forew the usefulness of clinical auscultation (Fig 1)

The fetal heart sounds appear to have been known at least as early as 1650 About that year a physician Philippe Le Goust (869), of Nior, France, wrote a poem making fun of his colleague

Mirac for claiming to hear the heart of the fetus "beating like the clapper of a mill" (615) The poem of Le Goust, written partly in Latin, partly in the Lamousin dialect, was republished by Philippeaux in modern French in 1879 (1206) Just as Parisinus by his asinine criticism of Harvey makes it clear that Harvey heard the heart sounds and that the phenomenon was not well known, Le Goust's equally ignorant satire leaves no doubt that Mirac discovered obstetrical auscultation

A number of references to murmurs can be

The Posthumous

# WORKS

OF

## ROBERT HOOKE, M D S R S

Geom Prof Gresh &c

Containing his

### Cutlerian Lectures,

AND OTHER

### DISCOURSES,

Read at the MEETINGS of the Illustrous

### ROYAL SOCIETY

IN WHICH

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III An Hypothetical Explication of MEMORY how the Organs made use of by the Mind in its Operation may be Mechanically understood

IV An Hypothesis and Explication of the cause of GRAVITY or GRAVITATION MAGNETISM &c

V Discourses of EARTHQUAKES their Causes and Effects and Histories of several to which are annexed Physicall Explanations of several of the Fables in Ovid's Metamorphoses very different from other Mythologicall Interpretations

VI Lectures for improving NAVIGATION and ASTRONOMY with the Descriptions of several new and useful Instruments and Correspondences the whole full of curious Disquisitions and Experiments

Illustrated with SCULPTURES

To these DISCOURSES is prefixt the AUTHOR'S LIFE giving an Account of his Studies and Employments, with an Enumeration of the many Experiments, Instruments Contrivances and Inventions by him made and produced a Catalogue of Experiments to the Royal Society

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FIG 1 Title page of the posthumous publication containing Hooke's reference to the heart sounds

found in writings between Harvey and Laennec. Some were the loud variety audible at a distance, others required application of the ear to the chest or other part. The following instances represent a sampling.

In 1674 Caspar Bartholin the Elder (1616-1680) wrote (1360) about "a young lady of Copenhagen" sometimes visited by a continuous headache. At these times she felt the pulse in the carotid arteries so violently in her head that a sound might be heard from a long distance like a clock.

Another early reference is that of Cornelius Stalpert van der Kolk who was born in 1620 and first published his book in 1667 (1433). Moragni (1114) quotes him as follows: "I once told me of a girl being cured by him who had her face puffed and very much swollen and had never undergone any menstrual purgation in whom he could very distinctly hear the agitation of the water in the pericardium when the heart was pulsing (for she laboured under a palpitation of the heart). This patient disorder was interpreted as hydro-pneumopericardium by Brucketeau (174) writing in 1844. However it may be that she had chronic nephritis with uræmia and that it was an ordinary albeit very loud, pericardial friction rub which was heard.

In 1713 James Douglas (1670-1742) of St Bartholomew's Hospital London made the following observation in a patient found at autopsy to have toxic regurgitation (964).

"Which is most incredible at sometimes the trembling, and the throbbing made with a noise in his Breast a plaint could be heard at some Distance from his Bed side."

Bouillaud (139) writes that Visconti long before Laennec discovered intussusception pointed out the more or less gurgling sound which is produced by the motion of gas into the cavities of the heart of animals. Pierre Hubert Vayer (1774-1818) a physician born in Belgium put an end to his career in 1811.

In 1757 the noted William Hunter (1718-1783) of London described in a case of arterio-venous fistula (varicocele aneurism) a thrill (particular vibratory movement) and a murmur (bruissement) which he compared to that produced by air in passing through a small

opening, or to the sound of the letter R spoken in a prolonged whisper (731).

In 1803 Benjamin Frerks (1781-1858) then a demonstrator in anatomy at Guy's Hospital London (1010), and surgeon to the East India Company reported (147) on an aneurism in an "isthmus" of the aorta. The patient had felt a sudden snap on the left side of her forehead. Thereafter she had a constant noise in her head which to her attention exactly resembled the blowing of a pair of bellows. The murmur increased when he lowered her head. Protrusion of the left eyeball developed as well as a throbbing tumor at the inner canthus. Frerks noted that when he occluded the left carotid the "trophism" diminished and the "distinct sound between a hiss and a buzz" likewise disappeared. He treated the left carotid artery with cure of the patient.

Writing on aneurism in 1812 (1274) Baron Antelme-Balthazar Richerand (1779-1840) of Paris described a case of aneurism in 1803 who sought exemption on the basis of an injury to an arm. An arterio-venous fistula was found. The ear applied to the bend of the elbow heard a faint murmur produced by the friction of the blood against the margins of the opening between the vein and the artery.

About 1817 Baron Larrey (1746-1842) who during the Napoleonic Wars assumed fame by his contributions to military medicine described the murmur of an arterio-venous fistula which had resulted from a saber wound. The clapper was cured by a considerable tumor which hid a pulsation synchronous with the pulse. Further more one felt and heard more deeply and in the direction of the pulsity vein a singular murmur (bruissement) like that which would be produced by a liquid which was made to pass through many crooked metallic pipes (812).

In 1815 in his *Treatise on Diseases of Arteries* (63) Joseph Hodgson (1768-1869) of Birmingham described a case of femoral arterio-venous fistula. The tumor presented a particular vibratory movement which caused a certain humming to be heard or rather a humming, corresponding to the arterial pulsation. The bruit was more distinct when the ear was placed on the tumor at the point where the sword had

penetrated. It was also readily audible 3 or 4 inches from this point."

Cardiac murmurs were less frequently noted, however. In 1806, in his famous *Essai sur les Maladies du Cœur*, Jean Nicholas Corvisart (1753-1821), physician to Napoleon and one of Laennec's teachers, wrote as follows about mitral stenosis (297): "Among the several specific signs which permit recognition of this affection is a particular murmur ('bruissement'), difficult to describe, which shows that the blood is passing through an orifice which is not large enough in proportion to the quantity of fluid to which it must give passage." The word "bruissement" used by pre-Laennec French writers for 'murmur' was also used for the "buzzing" (of bees), "rustling" (of leaves), "murmuring" (of brook), "rumbling," and so on. Corvisart also wrote as follows (298): "Some authors assert that they have been able to hear in certain cardiac maladies the sound produced by the beating of this viscus even at a great distance from the bed of the patient. I have never had the occasion to verify these observations. I have only heard the beatings when the ear was applied to the chest of the patient." However in 1826 Laennec (831a) stated: "The professor never put his ear to the chest."

The clearest descriptions of heart murmurs before Laennec are those of Allan Burns (1781-1813) of Glasgow (Fig. 2) who in his brief lifetime wrote what is essentially the first textbook



FIG. 3 René Thérèse Hyacinthe Laennec (1781-1826)

of cardiology in the English language. It is true that the murmurs he recorded were not of his own observation. He quoted (199 p. 187) a Dr. Brown and a Dr. Rutherford who had a patient with a hissing noise as of several currents meeting; the sound was frequently audible as in the varicose aneurism. On dissection the mitral valve was found indurated and reticulated. There seemed to be an opening left between the auricle and ventricle during the contraction of the latter. Thus on each contraction the blood flowed in part into the aorta, in part into the left auricle producing regurgitation along the pulmonary veins. The regurgitation of the blood from the ventricle into the auricle must have produced the hissing. Burns also quotes (199 p. 95) the description by a Dr. Dinton of a patient in whom "the violence of the palpitation was such that it was both audible and visible at a distance."

Laennec (Fig. 3) had made a consistent practice of direct auscultation for several years before he devised the stethoscope. He wrote (831a):

Byle [Gaspard Laurent B. 1774-1816, writer of a monograph on pulmonary tuberculosis] was the first whom I saw employ it when we followed the service of Corvisart together. It was obviously from considerable experience that Laennec (831b) wrote so technically about direct auscultation. As inconvenient for the physician as for the patient, distance alone renders it almost impracticable in the hospital; it cannot even be



FIG. 2 Silhouette Allan Burns (Courtesy of the Library, University of Glasgow)

proposed to most women and in most of them the volume of the breast a physical obstacle to its use. Baulle died the very year that the idea of the stethoscope came to Laennec.

I. J. Doublet of Paris, in 1817 (366) makes reference to immediate auscultation and seems to have claimed for himself its application to the study of respiration's sound. 'We must apply the ear closely to every point of all its [the chest's] processes by which means we can distinguish not merely the kind and degree of the sound but even its precise site.' I have frequently derived great benefit from this mode of investigation which is peculiar to myself and to which I was naturally led by the employment of the like method in exploring the pulsation of the heart. In 1811 (104) the British medical press had an amusing account of M. Doublet who is famous must be familiar to our readers as that of an eminent physician in Paris had lately occasion to read a memoir to the Academy of Sciences in which he mentions the following circumstances: having first directed his attention to the sound of the heart many years ago when he was taking leave of his mother he laid his head upon her bosom and wept in an agony of maternal grief at parting with him but her philosophic son was otherwise employed the while. He was struck with the distinct manner in which he heard the beating of her heart and the convulsive sob of her breathing—he listened to every sign as illustrating the principles of acoustics—and hence he ascertained the origin of the mode of examining into diseases of the chest by auscultation; now so generally adopted. M. Doublet evidently thought the anecdote redounded to his credit but we fear he will look in vain for any compliment on the score of feeling—truly his grandfather was indeed cold blooded.

Auenbrugger's description of percussion (1761) and especially Corvisart's translation (1809) of Auenbrugger's monograph popularizing the method were important in stimulating thought along lines of physical method for detecting internal disease. It was almost certain that a circumstance unrelated to the invention of the stethoscope that Corvisart was a teacher and colleague of Laennec. Observation had always been

practiced by astute clinicians and palpation was becoming more extensively practiced for example Morgagni (1114, vol. 1 p. 453) made reference to it as such. Furthermore in London Matthew Baulle (1761-1823) nephew of the brothers Hanter and the man to whom John Forster dedicated his translation of Laennec taught and practiced palpation a part of the physical examination including that of the heart. In fact for the practice Baulle was criticized by Sir Henry Hallford (1865) as treating his patients with unbecoming familiarity. Laennec in the famous passage which will be quoted later makes reference to the fact that both palpation and percussion were used in the examination before invention of the stethoscope.

The fetal heart sound was rediscovered by immediate auscultation before the stethoscope was invented or at least before publication of Laennec's monograph (1819). Laennec presented a preliminary report on the stethoscope to the Academy of Sciences in Paris in June 1818. On October 1 1818 Francois Marie Villot (1779-1844),urgeon of Geneva, read a paper before the Society of Physics and Natural History of that city and the following notice was recorded in the *Bibliothèque universelle* which was a Reader Digest of the arts and sciences (1033).

M. Villot believes he has discovered something of importance to the art of obstetrics namely a means of determining whether the infant is dead or alive in the mother's womb if the infant is alive but the pelvis contracted (caesarian operation) performed M. Villot has demonstrated that when one places the ear to the abdomen of the woman the beating of the heart of the infant are heard. No formal communications were published. However Villot made other verbal report in the following year. For example on July 8 1819 before the same society he reported use of direct auscultation in the diagnosis of twin pregnancies (1122).

## LAENNEC

René Theophile Hyacinthe Laennec (1751-1826) was a Breton who spent his professional career in Paris. Even without invention of the stethoscope he would deserve a place of honor in the annals of medicine for his pathological



studies, particularly those of cirrhosis, which bears his name, and of tuberculosis to which he himself succumbed at an early age. As pointed out above, Lennec was a prepared mind, as a result of the rediscovery of percussion by his teacher Corvisart and as a result of the practice of direct (immediate) auscultation by his good friend and colleague Robert Boyle.

Lennec's account of the discovery of the method of stethoscopy is probably the most famous single passage in medical literature (831c). "I was consulted in 1816 by a girl who presented the general symptoms of heart disease and in whom palpitation and percussion gave little information on account of the patient's obesity. Her age and sex forbade an examination [by direct auscultation]. Then I remembered a well known acoustic fact, that if the ear be applied to one end of a plank it is easy to hear a pin's scratching at the other end. I conceived the possibility of employing this property of matter in the present case. I took a quire of paper, rolled it very tight, and applied one end of the roll to the precordium, then inclining my ear to the other end, I was surprised and pleased to hear the beating of the heart much more clearly than if I had applied my ear directly to the chest."

Lennec's *Auscultation Méthode* first appeared in 1819. The three years following the first conception of stethoscopy in 1816 had been spent in intensive study of the method at the Necker Hospital in Paris and correlation of autopsy findings with clinical impressions. A second edition appeared in 1826, within weeks of his death.

Pulmonary disease in general and respiratory sound in particular clearly represented Lennec's "first love." He made several major errors in interpreting cardiovascular sound. He related the first sound ("bruit ventriculaire") to ventricular systole and the second sound ("bruit auriculaire") to atrial systole. Because of this confusion he never was able to relate murmurs accurately to lesions of particular heart valves. Lennec was not explicit as to how he thought contraction of the atrium and of the ventricle caused the heart sounds; probably he thought the contracting myocardium developed vibrations within itself. So often did autopsy reveal no cardiac lesion in patients in whom he heard a

murmur, that he concluded in his second edition that spasm of the heart or great vessels ("une contraction spasmodique du coeur") was responsible for most or even all murmurs, thereby abandoning the view expressed in his first edition that valvular obstruction produced murmurs and thrills. He never recognized the diagnostic significance of the pericardial friction rub even though Victor Collin, his *chef de clinique* (resident physician), gave a detailed description in 1824 in his doctorate thesis (280). ("There are few diseases more difficult to recognize than pericarditis. I must acknowledge that immediate auscultation does not afford certain signs of pericarditis.") He heard what we call venous hum and compared it to the sound of the "sea" or that produced by the application of a large seashell to the ears" but he thought the sound was arterial in origin. Corrigan (296) claimed that Lennec had no conception of the fact that murmurs and thrills are manifestations of one and the same vibratory phenomenon (see p. 43).

Lennec introduced the terms *bruit de soufflet* (bellows murmur, blowing murmur) and *bruit de lime* (bruit de rape) and *bruit de scie* (filing, rasping, and sawing murmurs, varieties of musical murmurs). It is likely that Lennec described the auscultatory sign of mediastinal emphysema which Humm (see p. 17) rediscovered over a century later. He referred to the clicking sounds as *le râle crépitant* and *le râle grosses bulles* (1370).

He wrote as follows: "Sometimes, although very rarely, it happens, in connection with palpitations that each contraction of the ventricle is followed by several successive contractions of the auricles which together do not occupy more time than in ordinary single contraction. Sometimes I have counted two pulsations of the auricles for one of the ventricles; other times four but the most frequent number of the successive contractions corresponding to one contraction of the ventricle was three. I have observed them only in persons affected by ventricular hypertrophy. Since Lennec considered the normal second sound to be related to contraction of the auricle, he was probably dealing in this passage with a variety of state in which one or more extra sounds occur at the end of systole.

or early in diastole (p. 166) like sy tolu click, plus second sound mitral opening nip early diastolic snap of constrictive pericarditis proto diastolic gallop Extra systoles may have accounted for some of his case. Clearly the constrictions of the auricle cannot be taken literally as have some authors who interpret the passage as a description of mitral heart sounds with heart block.

Laennec described noise and musical cardiac pulmonary murmurs. In certain persons the anterior borders of the lungs extend in front of the heart. If one examines such a person when his heart is beating more forcibly than usual the diastole of the heart compressing these portions of the lungs and forcing the air out of them alters the breath sound in such a way that it imitates a blowing murmur or the sound of a wood file. It disappears almost entirely when the patient is made to hold his breath for a few moments.<sup>2</sup>

Laennec (831) first invented the term *pneumo pericardium* and wrote as follows: 'Sometimes the air is combined with a liquid and this is by much the most frequent cause at other times the pericardium is distended by air alone. I have sometimes been enabled to announce its presence from the superintention of an increased resonance over the lower part of the sternum and from the existence of the sound of fluctuation produced by the action of the heart and by deep inspirations. I am convinced that in almost all the cases where the sound is heard at a distance the cause of the phenomenon is a temporary development of gas in the pericardium.'

The romance of Laennec's discovery and of his premature death from tuberculosis has captivated the imaginations of lay writers as well as of students of medicine and medical history. In 1919 *Docteur Laennec* a reasonably documentary motion picture account of his life was a hit in Paris. Kipling in a short story written in 1908 and entitled *Martiale Blanches* has Laennec captured by the British in the Napoleonic Wars and billeted in a small English village (150). One day the heroine comes upon Jerry the local medico and Laennec (the two are

the witches of the title) playing with toy trumpets. They were not real trumpets because Jerry put his trumpet against René's collar and listened while René breathed and coughed. Said Jerry 'This wonderfully like hearing a man's soul whispering in his inward but unlike I've a buzzing in my ears you make about the same kind of noise as old Coffer Markin—but not quite so loud as young Cooper. It sounds like the breakers in a reef—a long way off Comprehend? Perfectly, answered René. He knew the significance of the sound in his chest, and in his soul he said 'I drive on the breakers. But before I take I shall save hundred thousand millions perhaps by my little trumpet.'

### THE STETHOSCOPE

The well known 'cottonie flet' referred to by Laennec in the famous passage quoted earlier was known to Leonardo da Vinci (1452-1519) who described (1506) the forerunner of the stethoscope. 'If you cause your ship to stop and place one end of an ear in the water and the other end to your ear you will hear him at a great distance from you. You can also do the same by placing the end on the ground and you will then hear anyone passing at a distance from you. It is unlikely, however that da Vinci's observation was known to Laennec.

Laennec described his own stethoscope the model which quickly replaced the rolled up 'quirt of paper' in the following manner (831).

It consists simply of a cylinder of wood perforated in its centre longitudinally by a bore three lines in diameter and formed so as to come apart in the middle for the benefit of being more easily carried. One extremity of the cylinder is hollowed out into the form of a funnel to the depth of an inch and half which cavity can be obliterated at pleasure by a piece of wood so constructed as to fit it exactly with the exception of the central bore which is continued through it so as to render the instrument in all cases a pervious tube. The complete instrument—that is with the funnel hinged plug inserted—is used

<sup>1</sup> Quote 117 Hirschfelder (670) p. 114

<sup>2</sup> (Cf Figures 4 and 5)

in exploring the signs obtained through the medium of the voice and the action of the heart, the other modification, or with the stopper removed, is for examining the sounds communicated

by respiration." With his own hands Laennec built most of the first stethoscopes.

Although mild objections and a certain amount of ridicule accompanied the early years of the



FIG. 4. Rigid monaural stethoscope. (Unless otherwise specified exhibited and photographed through courtesy of Museum Armed Force Institute of Pathology, Washington, D. C.) (1) Replica of Laennec's quire of paper very tightly rolled (1816). (2) Invented by C. J. H. Williams (London, 1837). (3) Attributed to Sir William Ferguson (1809-1877), surgeon of Edinburgh (before 1863). (4) Combination stethoscope and percussion hammer said to have been invented by Salzer (before 1872). (5) Attributed to Heinrich Quincke (1842-1922), Kiel (before 1868). (6) Metal stethoscope of Linnard (1876). (7) Combination stethoscope and percussion hammer presumably invented by Felix von Niemeyer (before 1868). (8) Student's stethoscope, Zurich (1876), price 70¢. (9) Metal stethoscope attributed to Ludwig Fraube (before 1876). (10) Russian stethoscope captured in Korea (1950). (11) Replica of Laennec's wooden stethoscope (1819) (from Dr. W. I. Roger Sharp and Dohme, courtesy of Dr. Richard H. Shryock).

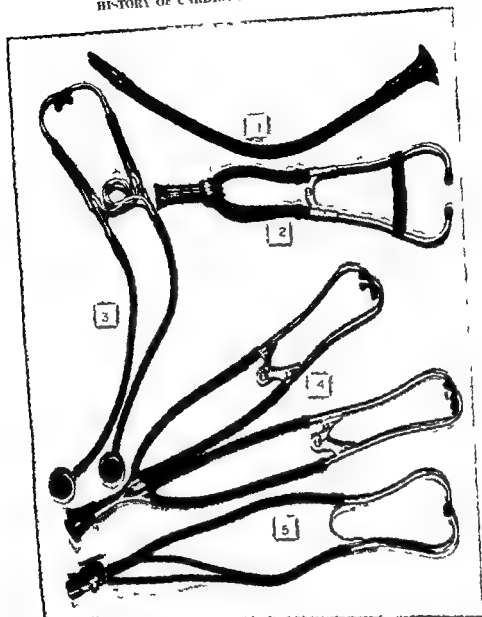


FIG. 5 Flexible stethoscope—most historical (Lancaster others) indicated exhibited through courtesy of Museum of Armed Forces Institute of Pathology (1) Type made as early as 1830 chest piece at right end (2) Invented by George F. Camman New York (1830) (3) Kerr umbilicophone (Courtesy of Dr Robert W. Carr, Baltimore) (4) Teaching stethoscope double (before 1896) (5) Modern stethoscope with Sprague Bowles combination chest piece

stethoscope in general its use became rapidly widespread with none of the period of oblivion that percussion suffered for over 30 years. Paris was then the leading medical center of the world and there were numerous young men studying in Paris who enthusiastically received the new clinical tool and carried it home with them to all parts of the world.

John Forbes (1767-1861) published an English translation of Laennec's book in London in 1821 and in Philadelphia in 1823. A German translation appeared in 1822 and an Italian translation in 1833.

In 1825 the year he graduated at Edinburgh (he and Corrigan were classmates) William Stokes (1804-1878) famous in connection with

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FIG. 4 Rigid monaural stethoscopes (Unless otherwise specified exhibited and photographed through courtesy of Museum Armed Forces Institute of Pathology Washington D. C.) (1) Replica of Iennec's "quire of paper very tightly rolled" (1816) (2) Invented by C. J. B. Williams (London (1837) (3) Attributed to Sir William Ferguson (1809-1877) surgeon of Edinburgh (before 1863) (4) Combination stethoscope and percussion hammer said to have been invented by Solger (before 1872) (5) Attributed to Heinrich Quincke (1812-1912) Kiel (before 1868) (6) Metal stethoscope of Inard (1876) (7) Combination stethoscope and percussion hammer presumably invented by Felix von Niemeyer (before 1869) (8) Student's stethoscope Zurich (1946) price 7 sh. (9) Metal stethoscope attributed to Ludwig Traube (before 1876) (10) Russian stethoscope captured in Korea (1944) (11) Replica of Iennec's wooden stethoscope (1819) (from Dr W. I. Boger Sharp and Dohme courtesy of Dr Richard H. Shryock)

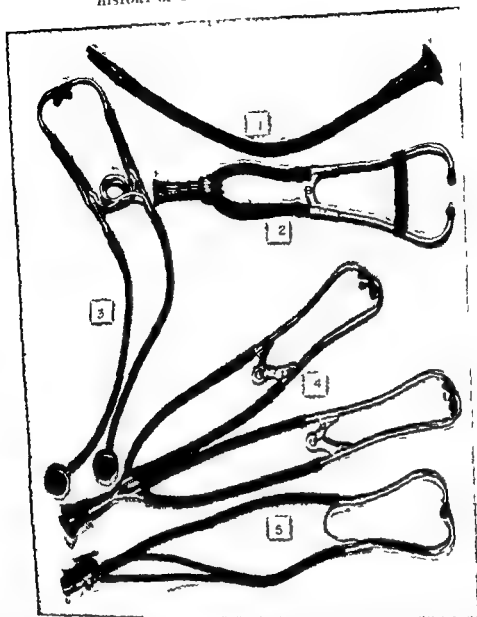


FIG 5 Flexible stethoscope mostly binaural (Lale's others) exhibited through courtesy of Museum of Armed Forces Institute of Pathology: (1) Type used as early as 1830 chest piece at right end (2) Invented by George I. Canham New York (1833) (3) Kerr sphygmophone (Courtesy of Dr Robert W. Carr Baltimore) (4) Teaching stethoscope double (before 1896) (5) Modern stethoscope with Sprague Howles combination chest piece

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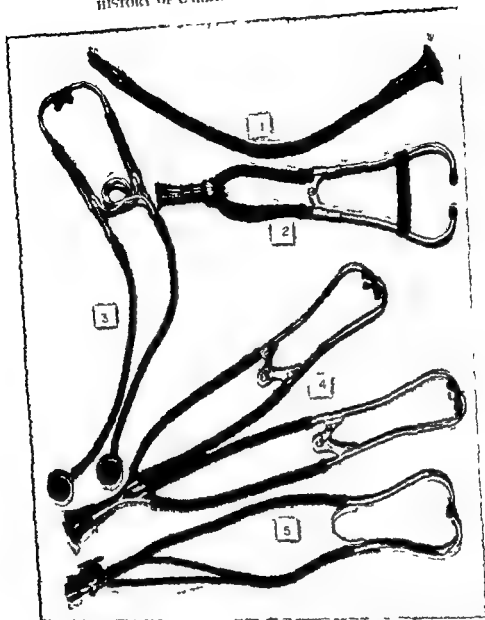


FIG. Flexible tetho-cope mostly binaural (Unless otherwise indicated exhibited through the courtesy of Museum of Armed Forces Institute of Pathology) (1) Type used as early as 1830 chest piece at right end (2) Invented by George I. Camman New York (1833) (3) Kerr sphygmophone (Courtesy of Dr. Robert W. Carr Baltimore) (4) Teaching stethoscope double (before 1896) (5) Modern stethoscope with Sprague Bowles combination chest-piece

tetho-cope in general its use became rapidly widespread with none of the period of oblivion that percussion suffered for over 30 years. Paris was then the leading medical center of the world and there were numerous young men studying in Paris who enthusiastically received the new clinical tool and carried it home with them to all parts of the world.

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In 1821 the year he graduated at Edinburgh (he and Corrigan were classmates) William Stokes (1801-1878) famous in connection with



heart block and periodic breathing (also see p. 22), published a small book entitled *On the Use of the Stethoscope*.

Sir Charles Scudmore (1779-1849) of London returned from Paris with his initial reservations about the stethoscope changed into enthusiasm when Laennec, discovering that Scudmore's ear had an unusually large trigus, hid the end of the stethoscope hollowed out to obtain a better seal (1865). In the clinic of Nasse (1778-1811), whose name is associated with the law describing the sex-linked inheritance of hemophilia, the stethoscope was used early in Germany. The large group of American students in Paris in the first half of the last century brought the gospel back with them. W. W. Gerhard (1809-1872) of Philadelphia wrote a comprehensive text on percussion and auscultation. Henry Ingraham Bowditch (1808-1892), uncle of Henry Pickering Bowditch (1840-1911), the Harvard physiologist who discovered the "all or none" law of heart, wrote a manual called *The Young Stethoscopist* (1846), and Oliver Wendell Holmes (1809-1894) wrote both a prize-winning Boston essay (1836) and a satirical poem (p. 52) on the new method.

An interesting view of the reception of the stethoscope in London is provided by the *London Times* which on December 19, 1824, carried the following piece (1110):

'A wonderful instrument called the Stethoscope invented a few months ago for the purpose of ascertaining the different stages of pulmonary affections is now in complete vogue in Paris. It is merely a hollow wooden tube about a foot in length (a common flute with the holes stopped and the top open would do perhaps just as well). One end is applied to the breast of the patient, the other to the ear of the physician, and according to the different sounds harsh, hollow, soft, loud, etc. he judges of the state of the disease. It is quite a fashion if a person complains of a cough to have recourse to the miraculous tube, which, however cannot effect a cure, but should you unfortunately perceive in the countenance of the Doctor, that he fancies certain symptoms exist it is very likely that a nervous person might become seriously indisposed and convert the supposition into reality."

It is not improbable that the comment of the *London Times* was influenced by John Forbes' mistaken, indeed silly, evaluation in the translator's preface to the 1821 English edition of Laennec (468):

That it will ever come into general use notwithstanding its value I am extremely doubtful because its beneficial application requires much time and gives a good bit of trouble both to the patient and the practitioner, because its whole hue and character are foreign and opposed to all our habits and associations. It must be confessed that there is something even ludicrous in the picture of a grave physician proudly listening through a long tube applied to the patient's thorax as if the disease were a living being that it could communicate its condition to sense within.

Pierre Adolphe Piorry (1794-1870) who advocated the use of a pleximeter (or pleximeter) in percussion so called *mediate percussion* "sclanderized" the stethoscope by reducing its diameter to that of a finger (1211). He also introduced the trumpet chest piece and improved the ear piece so that better seal was attained (1243). He introduced a removable ear piece for greater portability. His was the stethoscope used in France—when my was used—for the next 75 to 80 years.

C. J. B. Williams (1803-1889) is cited by his son (1963) to have determined that a flexible stethoscope was as satisfactory as the solid type. In fact in the 1830's Williams stated the following in an article for the *Cyclopedia of Practical Medicine*:

The flexible caoutchouc tube terminating in a small ivory funnel now in common use by deaf persons is employed by some auscultators and in some respects answers the purpose well enough but it is in others decidedly inferior to the solid instrument.

In 1839 Chiracilly of Tours (250) in passing, pointed out the advantage of the flexible stethoscope in permitting simultaneous observation of the pulsations in the neck and auscultation of the precordium. It was in the analysis of what we would now call diastolic gallops that Chiracilly found it useful.

In *The Young Stethoscopist* (1816) Bowditch refers to the monaural flexible stethoscope used by Caspar Wistar Pennock of Philadelphia. In 1814 Charles Goodye developed the method

for improving rubber by addition of sulfur to the native product. This technologic development paved the way for the flexible tethoscope.

American physicians can take pride in the fact that the binaural flexible stethoscope originated in this country. George P. Cimmarrn (1804-1863) is usually credited with its invention. By 1832 he had already developed a stethoscope with flexible tubing (spiral of wire covered with silk later rubber or as he called it "caoutchouc") with ear pieces and a spring iron piece to hold the ear pieces in place. The formal announcement of this stethoscope appeared in 1833 (223). In 1861 S. S. Alston in his *Physical Examination of the Chest* (p. 324) pictured the Cimmarrn stethoscope which is almost precisely like the modern binaural stethoscope with bell chest piece.

Curiously enough in France Laennec's homeland the tethoscope was slow in gaining wide use and direct auscultation was generally practiced instead of stethoscopy. But who was prominent in introducing the binaural stethoscope into France is quoted (187) as making the following statement about 1933: "Until twenty five years ago immediate auscultation was used almost exclusively in France. The binaural stethoscope was absolutely unknown. The monaural was on the table of every practitioner but seldom used being so utterly impractical. I was converted to the use of the binaural tethoscope on my first journey to the United States in 1908 and have demonstrated it to my students since that time while my colleagues looked at me with awe and thought I was making myself ridiculous." In 1936 Froese or Étienne Bernard (91) wrote that up until a relatively recent date most practitioners in France auscultated patients by applying the ear directly to a thin handkerchief placed on the chest of the patient. However since a time which can be fixed at about 20 to 25 years ago use of the binaural stethoscope has become more and more widespread and it can be said that at present most practitioners use it. It was the advent of the auscultatory method for measuring blood pressure which made the binaural tethoscope unavoidable.

The important studies of the giant of French

cardiology such as Duroziez (see p. 27) and Potain (see p. 21) were done by direct auscultation or by means of the monaural stethoscope. In 1907 Charles Laubry (see p. 26) wrote me that he never saw his old chief Vaquez use a binaural tethoscope until about 1913 or 1916.

The combination of the prevailing practice of direct auscultation by the French profession with the Frenchman's particular brand of humor is doubtless responsible for numerous cartoons satirizing physicians (see Fig. 20).

There have always been those who champion the superiority of the naked ear at least for some varieties of sound. Lewis A. Connor (1866-1930) of New York City, a founder of the American Heart Association and one of the first editors of the American Heart Journal pointed out that faint high pitched aortic diastolic murmurs are sometimes better heard by direct auscultation than by stethoscopy (252). He is said always to have carried a silk handkerchief for use in direct auscultation.

There have also been those who preferred the monaural solid stethoscope to the flexible binaural instrument. Writing in *Lancet* in 1902 (1417) one Syer stated: "I look upon the use of the binaural stethoscope as being in every way most objectionable. The double stethoscope should be altogether done away with and abolished from the face of the earth—Disend it—Cartago. To this very day there are many parts of the world including most of central Europe where the monaural tethoscope is used. It is the teaching in the medical schools in these parts that one hears too much with the binaural stethoscope and is likely to be confused thereby. Pediatricians are usually the only ones to use the binaural flexible stethoscope the advantages being that it is possible to follow the movements of the child and extraneous noise is better excluded. It is interesting to find the same evaluation of the relative merits of the two types of stethoscope in Simon's *Physical Diagnosis of the Heart* 1881 (p. 92).

The next main development in the direct physiognomy of the modern tethoscope was the diaphragm (or membrane) chest piece usually attributed (187) to R. C. M. Bowles, an engineer of Brookline, Massachusetts who patented the

diaphragm in 1894. When it was recognized that both the bell and the diaphragm have merit and in effect complement each other, a composite chest piece permitting rapid change from one to the other by means of a valve was the next advance. In 1926 Howard B. Sprague (189-) of the Massachusetts General Hospital, Boston, introduced the combination chest piece (1428) now in general use.

Studies of stethoscope function have been conducted by several workers, particularly Rappaport and Sprague (1244, 1245), and have defined proper length and bore of tubing (1243), the importance of snug fit of the ear pieces (1242), the distinctive function of the bell and diaphragmatic chest pieces (1245), the proper structure for these chest pieces.

Several models and variations of the stethoscope have not survived. In 1859 Alison (1c) invented a stethoscope in which the chest piece was a small vat of water. He suggested that improved coupling with the chest, including close fit between ribs, might improve audibility of chest sounds. The phonendoscope (99) invented by Buzzi, a physician, and Bianchi, a physician, of Parma, Italy, in the 1890's is another example of an obsolete stethoscope (Fig. 6 A and B). A small knob on the end of a rod was applied to the chest. The other end of the knob was attached to the center of a diaphragm. A second diaphragm was mounted parallel to the first with only a small air space between the two. The second diaphragm was comparable to the ordinary single diaphragm chest piece. The ad-

vantages of the phonendoscope were said to be precise localization of sounds and simplification of faint sounds. The idea of a stiff diaphragm is, perhaps, the only surviving feature of the phonendoscope. Parker's refractoscope (1181) made use of a resonating cavity whose volume could be varied.

In 1884, one Aydon Smith (140c) quite seriously recommended an instrument which could be used as a manural, buural, or differential stethoscope, the tubing could serve as a tourniquet, either for stomach tube, and the chest pieces as otoscopic speculum or funnel for administering fluids through a tube. Enemas or douches could also be administered.

Differential stethoscopes of various types were designed. In 1861 Alison discussed those in existence at that time (16). The principal differential stethoscope designed in recent years is that of Dr. William Kerr (1889-) of San Francisco (formerly Professor of Medicine at the University of California (78c)). The Kerr sphygmophone (Fig. 5) is a double stethoscope in which chest pieces are concerned. Sounds picked up by each chest piece are led into both ears. The advantage is said to be ease of comparing the sounds in two areas of the precordium or of the lung fields.

The electric stethoscope for multiple student teaching and for aid to the deaf physician was a logical development paralleling those in the field of hearing aids, telephony, and electronics. Cribot's dating from the early 1920's is one of the first examples (206).

The stethoscope has, of course, become virtually



FIG. 6 The phonendoscope of Buzzi and Bianchi (from Bianchi's monograph (93))

the symbol of the medical profession Dr Arthur Conan Doyle (1859-1930) has his Sherlock Holmes make indirect use of the stethoscope in identification

As to your practice if a gentleman walk into my room smelling of iodiform with a black mark of nitrate of silver upon his right forehead and a bulge on the side of his top-hat to show where he has secreted his stethoscope I must be dull indeed if I do not pronounce him to be an active member of the medical profession

—A Scandal in Bohemia Chapter 1

### THE GOLDEN CENTURY OF STETHOSCOPY (1819-1919)

There is nothing so captivating as new knowledge. Even though its subject be incurable diseases which it renders not a whit the less incurable till it is captivating. Cases of such diseases [i.e. with murmurs] always abounded in hospital. They were essentially difficult cases. Their symptoms were hard to interpret into any definite meaning. They betokened that in some way or other the heart was diseased and that in some way or other their termination was death. Day by day to watch over these cases and to treat them was an irksome duty—it was even a thing to damp the spirit.

But auscultation brought to them a new light and a new interest. And then these cases became the cases which we were continually busy about which we were never tired of visiting and examining and auscultating and of examining and auscultating again and again.

—Latham Lectures 1947 (1943) p. 231

The golden century of stethoscopy began with Laennec and can be said to have ended with Sir James Mackenzie. It was the age of Boullaud, Hope, Flint, Durosoir and Potain to mention only five among many. It was a period of exciting exploration and occasionally extravagant claim. Virtually all the phenomenology of cardiovascular sound was described in this period. It can be considered to have ended in 1908 with the first edition of Mackenzie's *Diseases of the Heart*. It ended then in part because Mackenzie de-emphasized valvular disease *per se* and deemed the grave prognostic significance that was then usually attached to any murmur in part because about 1908 the age of stethoscopy was replaced by the age of Phonocardiography. Since 1908 virtually all studies of cardiovascular sound have had phonocardiographic documentation although a few strictly stethoscopic studies of importance have continued to appear.

The several workers in the golden century of stethoscopy and their contributions will be considered in alphabetical order. Some liberty with time has been taken inasmuch as two or three persons whose contributions were made later than a century after Laennec are included.

Jean Baptiste Boullaud (1797-1881) Paris (Fig. 7) wrote extensively on cardiovascular sound in his *Traité Clinique des Maladies du Cœur* of which the first edition appeared in 1831. He added (1924 p. 14 ff) to have described and named *bruit de galop* (gallop rhythm) in 1847. He assigned the designation *bruit de rappel* (a particular type of drum beat) to a group of conditions including *split second sound* and *mitral opening snap*. He also compared the sound (140 p. 213) to that of the hammer which after striking the iron rail on the road rebounded and fell again motionless. Boullaud described venous hum under the designation *bruit de diable* (diablo being the name of a French toy like a humming top). He noted its frequent occurrence with menarche. He described the musical type of venous hum and used musical notation to represent examples in his textbook (140 p. 264). However like Laennec he assigned its origin to the carotid arteries. Interestingly he called the musical venous hum *chant des artères*.

The following page is selected from among Boullaud's many observations on cardiovascular sound (140 p. 187). I have encountered in large number of cases where the valvular sounds had a timbre so dry, so snapping and so harsh that one would think he was hearing the sound which two sheets of parchment produce by striking each other abruptly and forcefully hence the name *rip* (cliquement) or parchment sound (*bruit de parchemin*) by which I have been in the habit of designating this modification of the valvular sound. Boullaud related his parchment sound to fibrosis in the valve. Boullaud also described a quadruple movement and quadruple heart sound *rhythme à quatre*.

\* Writing in 1884 Alfonso Clark (1807-1885) humorously stated that a friend of his translated this freely as 'a devil of a noise' (1977).

\* The German called it *Vonnensquern* (nun's murmur) possibly because it is more frequent in the elderly trusting and notorious or undernourished females.



FIG. 7 (Upper left) Bouillaud (upper right) Fauvel (lower left) Hope (lower right) Hope's own drawing of his case of aneurysm of the aortic sinus of Valvula ruptured into the right ventricle. From 1839 (3rd) edition of his *Diseases of the Heart*

temps in which the two intermediate sounds follow each other in quick succession and resemble a small dry crack." The basis was, he suggested, "a large, broad, calcified plaque forming an embossment on the surface of the heart" found at autopsy.

Richard C. Cabot (1868-1939) Boston described (with Locke) in three cases of intense anemia a diastolic murmur which was loudest at the fourth left costal cartilage and suggested aortic regurgitation (209). But at autopsy in all four the aortic valve was sound. Later Cabot

and colleagues made pioneer studies of the frequency composition of heart and lung sounds (208) The first edition of Cabot's *Physical Diagnosis* appeared in 1900

In 1832 *Dominic John Corrigan* (1802-1880) of Dublin wrote on "permanent patency of the mouth of the aorta or inadequacy of the aortic valve" (293) The characteristic quality of the pulse since called by his name was pointed out and the murmur of aortic regurgitation was partially described His pioneer studies on the mechanism of murmurs and thrills (296) were described on page 43

*John Elliotson* (1791-1868) of London first professor of medicine University College (1823) was an imaginative person a minor genius and a true observer He called attention to Robert Hooke's reference to the heart sound and prediction of tetothops In the *Lambert lectures* of 1840 he described four types of musical murmurs "I have heard it [the musical murmur] exactly resembling the cooing of a dove—a variety not mentioned I believe by authors" He discovered and fostered James Hope Later Elliotson lost practice and prestige because of his work with hypnosis and the treatment of hysteria He was one of the first to do surgical procedures under hypnosis and he was undoubtedly ahead of his time but the methods he used for publicity turned the profession against him

*John D. Fisher* (1797-1850) of Boston described cephalic murmurs (460)

*Louis Galliard* (1877- ) Lyons cardiologist (487) wrote about "a tole clicks" referring to the phenomenon as *bruit de triquet* and presented evidence for their origin in pleuropericardial adhesions (514) In calcific aortic stenosis he (521) described the dissociation between a noisy murmur in the aortic area and a musical murmur at the left femoral border and apex He (514, 515) emphasized the simulation of mitral stenosis where a late systolic click occurs He (511) called the sounds audible with atrial systole in heart block *galop de bloc* believing them due not to atrial contraction itself but rather to reperfusion in the ventricle of the atrial contraction A son Leon Galliard was working particularly in collaboration with Roger

Froment (488-491, 494, 495) has made contribution to phonocardiography

*Robert James Graves* (1797-1853) of Dublin helped establish the friction rub as a sign of acute pericarditis (586) He also described musical pericardial friction sound which he said resembled the sound of a wet finger rubbing on glass He probably described the benign though loud and weird-sounding musical murmur of pericardial origin heard sometime especially in young persons with febrile illness and sometimes persisting for months or years (patient Mary Robin on page 926)

Graves in describing the condition which bears his name stated that at times the accentuation of the heart sound in thyrotoxicosis is so marked as to render the first sound audible at a distance from the chest

I have lately seen three cases of violent and long continued palpitations in females in each of which the same peculiarity presented itself viz enlargement of the thyroid gland the size of the gland at all times considerably greater than natural was subject to remarkable variations in every one of these patients The palpitations have in all been considerably more than a fever and with such violence as to be at times exceedingly distressing and yet there seems no certain grounds for concluding that organic disease of the heart exists In one the beating of the heart could be heard during the paroxysm at some distance from the bed a phenomenon I had never before witnessed and which strongly excited my attention and curiosity She herself her friends and Dr Harvey all testified the frequency of this occurrence and said that the sound was at times much louder than when I examined the patient and yet I could distinctly hear the heart beating when my ear was distant at least four feet from her chest! It was the first or dull sound which was thus audible

The observation recalls Bartholin's observation (p 5) It also brings to mind a veterinary experience A cow developed angioneurotic edema with laryngeal edema and probably asthma After intravenous injection of what seemed an enormous dose of an adrenaline like material the respiratory distress was promptly relieved but the heart sounds were clearly audible at a distance of several feet for about fifteen minutes

*Louis Virgil Hamman* (1877-1946) of Baltimore described the aortic diastolic murmur which may accompany dissecting aneurysm of the aorta and attributed it to distortion of the aortic

ring by the hemistoma of the media (628) This sign had been described by Letulle in France in 1905 (884) and first in the English language literature in 1926 by William H. Reesnik and Chester S. Keefer (1262), then assistant resident physicians at the Johns Hopkins Hospital The latter writers thought, however, that the phenomenon was due to backflow of blood through the false channel In the introduction to the clinical pathological conference in which Hamman emphasized the sign of aortic regurgitation in dissecting aneurysm, he gave his charmingly expressed appraisal of the "C P C" as a teaching exercise (628)

It seems to me a delightful and entertaining exercise and one not altogether without profit As a method of instruction it has great advantages over the bedside clinic not, it is sure, to the physician discussing the problem, but certainly to his hearers At the bedside an able and experienced physician supported by the weight of reputation may make almost what he pleases of the clinical facts before him None will be so rash as openly to dispute his conclusions and nearly all will go away convinced of his skill and erudition But his position at a clinical pathological conference is quite different Here all the advantage is on the side of the hearer for though the physician may be supported by a reputation to equal O'Leary's and by the honors of all the Academies there sits the smiling pathologist ready and sometimes even eager to administer the *coup de grace* to his reputation erudition and eloquence What he may say is to be judged immediately and irrevocably He is no longer the glorious high priest of medical science introducing novices to her mysteries but the very humblest suppliant prostrate at her feet

Hamman also wrote about spontaneous mediastinal emphysema (Hamman's disease) and the adventitious sounds which are associated with it (630) (631) (632)

In 1828 Thomas Hodgkin (1798-1866) of Guy's Hospital London, and Hodgkin's disease fame described (694) a musical murmur in association with what he called retroversion of an aortic cusp and further defined as a particular state of the valves of the aorta, which by admitting of their falling back towards the ventricle unfits them for the performance of their function He called the murmur *bruit de scie*—a purring, thrilling, or sawing kind of noise" Hodgkin himself attributed the original observation to Kay

(1827) and the phenomenon in question is sometimes called the Hodgkin Key murmur

In his short lifetime (738A) James Hope (1801-1841) made many important contributions to the clinical phenomenology of cardiovascular sound and did animal experiments as well (Fig 7) His personality was marred by contentiousness of pathological proportions and a concern about priorities which amounted to paranoia In particular, C J B Williams (p 23) and Boullaud seem to have been objects of Hope's antipathy Hope's textbook *Diseases of the Heart* is perhaps the first English text of cardiology in the modern sense The first edition appeared in 1832, the third edition (1839) is particularly noteworthy and is both interesting and instructive to the modern reader, although as Litham commented in 1847 "its style, which is too often controversial, and even disputatious, repels many readers, and has been in some measure a hindrance to its usefulness" (711, p 470) For example, in the preface of the 1839 edition he wrote "I have ventured to reclaim for my countrymen and self a number of discoveries which an eminent French writer [probably Boullaud], probably from unacquaintance with the English language and medical literature, has imagined to have emanated from himself" When he performed experiments he recorded with utmost care the precise date and the name of the individuals who observed them he would even provide each observer with a protocol of the experiments to be performed mainly so that there could be no question about whose were the ideas behind the experiment

Hope invented the term 'venous hum' and supported the contention of Ogier Ward of Birmingham that the sound arises in the jugular veins not the carotid arteries as held by Laennec and Boullaud By the management of pressure with the stethoscope over or near large veins the venous murmur may often be raised by a gradual swelling into a more or less musical hum such as is yielded by a child's humming top I propose to denominate this Venous Hum for without being unnecessarily quaintish I think that this is not only a rather more euphonic epithet but more intelligible than noise of the devil by which term derived from a

playing (purring top) known to Sen M Bonilland has designated the hum in question (711 p 118)

Hope described the continuous murmur of aortopulmonary communication in a case of rupture of an aortic aneurysm into the pulmonary artery. It appears that a continuous murmur extended from the first over the second sound (711 p 470) He found a similar murmur with rupture of a aneurysm into the right ventricle. The last condition was also treated with a drawing by Hope himself (fig 7)

Hope described the cardiopulmonary murmur in two students of University College. Both wore very tight waistcoats preventing the expansion of the lower rib. During this state of breath-taking a light bellows murmur with the first sound over the semilunar valves existed in both. It was not however exactly synchronous with this sound but began an instant later as if from a separate cause. In both the murmur ceased entirely when unbuttoning their waistcoats and when blind, of their trousers they breathed with the lungs naturally inflated. By alternately the circumference the murmur could be created or removed at pleasure. I presume therefore that it proceeded from a cause exterior to the heart (711 p 391) Movement of air in compressed lung was suggested as the mechanism of the murmur

In discussing musical murmurs Hope wrote: The musical tone was a clear note like the coo of a pigeon and also ringing a semitone in the middle like the mew of a kitten. It attended the second sound and proceeded from aortic regurgitation (711 p 87) Hope noted the role of a vibrant member in the production of musical murmur — presentation of an edge to a tremor to be calculated to produce musical vibration. He also made the analogy to various musical tones — there is but a shade of difference in the mechanism by which we make the lips produce a blow or a whistle the latter depending on the happy and ready adaptation of the use of the aperture to the strength of the current.

Hope produced hoarse murmurs in dogs by copious bleeding. What was known as Hope's early diastolic murmur was probably the Graham-Steell murmur (see p 30)

An anonymous poet inscribed the following verse in a copy of Hope's textbook in the library of the Royal College of Physicians London (1164)

He opened wide the portals of the heart  
And bade us hearken to its varied part  
Taught us the rules of thescopic art  
And healed disease long running in its part

Henri Huchard (1844-1910) of Paris described the musical murmur which may be associated with aberrant tendon of the left ventricle (715-718) aortic heart sounds (717) with complete heart block (*égales en écho*) and variation of the first heart sound in complete heart block (717) with occasional very loud sound (*bruit de canon*). The clinical picture of aberrant tendon of the ventricle was described on the history of a 49-year-old patient with nephritis and cardiac hypertrophy. As well as a musical systolic murmur there was a presystolic gallop. At autopsy there was other than left ventricular dilation and hypertrophy, no lesion except in aberrant tendon (see page 184). Presumably the aberrant tendon is a congenital malformation giving no clue to its presence until dilatation of the ventricle pulled it out so that it vibrated musically during ventricular ejection.

In one patient a 70-year-old hypertensive with no peripheral signs of aortic regurgitation Huchard (716) described an exceedingly loud musical diastolic murmur which immediately suggested to observers including Duroziez rupture of a valve of the aorta. No valvular lesion was discovered at necropsy. Huchard himself thought the murmur probably aorticopulmonary. Percardial origin is also possible. Let it likely be a very light but not less at the aortic valve.

In his lectures on heart disease (1847) *John Vere Latham* (1789-1873) of St Bartholomew's Hospital London a magnificent clinical teacher organized well the practical aspect of instruction. His superb lectures were rendered memorable by the frequent use of epigram, bon mots, aphorisms and well expressed common sense.

Some of his contemporaries like competent and less discreet may have overdone the embellishments in lecturing and ward rounding and



even Iatham's approach may have been unattractive to some. In 1833 Henry Ingersoll Bowditch (see p 12), then aged 25 years and fresh from the service of Louis and others in Paris, wrote as follows about his visit in London "I followed Dr \_\_\_\_\_, one of the chief physicians of London, in his visit to the hospital. His main object seemed to be to make the students laugh. I was completely disgusted. Such is the case with most of them. They talk much but know little" (144).

In discussing factors determining the intensity of murmurs, Iatham pointed out (813, p 37) the usually great intensity of musical murmurs, remarking on a peculiar quality of the endocardial murmur, giving it a high musical note. Such a murmur will sometimes refuse to suffer restriction to a certain space within the body. It will even carry itself outwards and reach the ears of bystanders at a short distance." Iatham was instrumental in the introduction of English words such as *murmur* in place of the French equivalents such as *bruit de souffle*.

Jean Alexander Lejeune de Kergaradee (1787-1877), Paris, made stethoscopic studies of fetal heart sounds, uterine souffle, and other acoustic phenomena of pregnancy. The findings were published in 1822 in Magendie's *Journal de physiologie experimentale et pathologique* (872). Because of this excellent report Kergaradee, rather than Mayor (see p 7) long, received credit for discovery of the fetal heart sounds. It is in fact likely that Mayor's observations were unknown to Kergaradee and certainly Kergaradee was the person who placed the acoustic phenomena of pregnancy at the disposal of the profession (24).

Samuel A. Leane (1891- ) of the Peter Bent Brigham Hospital, Boston, has been a leading teacher of cardiac auscultation. In 1933 with Freeman (481) he suggested a system for the clinical grading of the intensity of murmurs. He has emphasized the relation between PR interval and intensity of the first heart sound and the usefulness of auscultation in the diagnosis of arrhythmias. He collaborated with W. P. Harvey in *Clinical Auscultation of the Heart* (1949).

Sir James MacKenzie (1853-1925) of Burnley

London and St Andrews (Fig 10), pointed out that the presystolic murmur of mitral stenosis disappears when the cardiac rhythm becomes totally irregular (1011). He stated that an explanation for this first occurred to him in 1891. He referred to auricular fibrillation as nodal rhythm because he found from recordings of the venous pulse and other beats that there was no evidence of atrial activity in such cases—he assumed that the pacemaker had moved to the atrioventricular node. At any rate, he is given the correct explanation to the disappearance of the presystolic murmur, namely that atrial systole was no longer occurring and that the atrium was in effect paralyzed.

MacKenzie emphasized the importance of the state of the myocardium as opposed to valvular disease. He derided the grim prognoses which at that time were often rendered on the basis of murmurs alone. In particular he emphasized the benignity of the apical systolic murmur when it occurred as an isolated finding.

The main contributions of Thomas Beall Peacock (1812-1882) of St Thomas' Hospital, London (Fig 8), were descriptions of the findings of necropsy which corresponded to an autopsy findings in life. He wrote two monographs—*Valvular Disease of the Heart* (1865) and *Malformations of the Human Heart* (1858 and 1866)—and contributed many articles to the Transactions of the Pathological Society of London.

In 1854 Peacock described a musical diastolic murmur in a 64-year-old patient (1194). It exactly resembled the sound produced by the common cuckoo clock and was so loud as to be heard at a distance of several feet. The aortic cusps were thickened and separated with retroversion of the free edge of the right cusp. Although the valve lesion may have been syphilitic, the double systolic diastolic character of the musical murmur and the relatively advanced age of the patient suggests atherosclerotic disease of the valve (See p 232 for discussion and Fig 273 for illustration of a probably similar cuckoo murmur of calcific aortic valve disease). In 1863 Peacock (1196) reported on a large number of patients with dissecting aneurysm of the aorta. Although he was one of the first to recognize that survival for years occurred in some patients



FIG. 8 (Upper left) Lotain (from biography by Tisser) (upper right) Durosoz (lower left) Tisser (lower right) Leacock

he did not note aortic regurgitation in any (cf p 452) Leacock (119, p 113) recognized the murmur of congenital pulmonary stenosis.

Throughout his career Pierre Carl Edouard Fétain (1825-1901) of the Charité Paris (Fig. 8) devoted his major attention to the clinical study of cardiovascular sound. His doctorate thesis in 1851 was devoted to the subject of the abnormal vascular bruit which follows hemorrhages (1227). His studies of gallop published in 1870 were outstanding (1225). He studied the presystolic gallop in particular referring to it as false reduplication of the first sound

and indicated its association with contracted kidney—the basis for the designation *bruit de brightique*. He described the gallop in this manner:

The sound is much duller than the normal sound; it is a hock, a distinct pulsation, scarcely a sound. When the ear is applied to the chest it affects the sense of touch more perhaps than the sense of hearing and on a more flexible stethoscope it nearly always disappears altogether. Potain held to the view that gallops are thus resultant from sudden filling of the ventricle.

*Nierengalopp* (kidney gallop) was the German term.

He expounded the important concept that gallop sounds are an exaggeration of normal elements. 'If one auscultates a goodly number of healthy persons, one will not be long in discovering that diastole is not always absolutely silent but in the part of the cardiac cycle where the normal sound responsible for the gallop is located, there is sometimes already, in vestigial form, something which, when exaggerated, could become the sound in question.' Dock (357, p. 36) wrote as follows:

Potain and his pupil made the French and Spanish speaking doctors acutely aware of the sounds in diastole which occurred with failure of the myocardium but in the English speaking world these sounds were either ignored or passed over lightly. Murmurs were the abnormalities sought. The *Oster* of 1920 has galloping conumption but not gallop rhythm in its index.

Although Schifer is said to have pointed it out in 1858 (1355), Potain (1226) in 1866 clearly described normal inspiratory splitting of the second heart sound. He described the early systolic click and attributed it to distension of the pulmonary artery or aortic the mid-systolic click and attributed it to pleuropericardial adhesions. Potain assigned the term *bruit de labour* to the ringing aortic second sound in conditions such as luetic aortitis.

In 1856 (1223) Potain described the early diastolic snapping sound of constrictive pericarditis. 'The second sound was reduplicated or rather composed of two sounds following one another in quick succession one clear dry ringing, with the usual characteristics of a valvular snap and of more or less unchanging intensity the other coming immediately after words less well defined less resonant coinciding exactly with the impulse felt in the precordial region, like the impulse increasing during inspiration, and taking on at that point a rather metallic tone.' The "impulse is the *Spitzenstoss* of Skoda (1390), translated as diastolic heart beat" by Francis C. Wood of Philadelphia (1587) a characteristic of constrictive pericarditis.

Potain (1224) maintained that all circumscribed systolic murmurs be they early mid or late

systolic, are non organic as compared to the murmur of mitral regurgitation which is holo systolic.

Potain is also credited with invention of the dilution pipette for blood cell counts and of a sphygmomanometer with which he made pioneer observations on hypertension. About 1867 he attempted to record the heart sounds by means of tambours but discovered, of course, that the vibrations are of too low amplitude.

Josef Škoda (1803-1881) actually Škodý, pronounced Schkodý, Professor of Medicine in Vienna, was a close clinical collaborator of the pathologist Rokitsky (a Czech like himself) at the Allgemeine Krankenhaus. Škodý was born in Pílen, Czechoslovakia where his brother Johann founded the well known Škodý works. The first edition of Škodý's textbook on percussion and auscultation appeared in 1839. Škodý emphasized the frequent occurrence of faint aortic heart sounds and accentuated pulmonary second sound in mitral stenosis—sometimes in the first called Škodý's sign (1390).

George F. Still (1868-1941) pediatrician of the Great Ormond St. Hospital London, for whom Still's disease is named, described a musical variety of functional murmur in children often known as the twanging string murmur (1446).

And here I should like to draw attention to a particular bruit which has somewhat of a musical character but is neither of sinister omen nor does it indicate endocarditis of any sort. In my own notebooks I am in the habit of labelling it physiological bruit but only for want of some better name. It is heard usually just below the level of the nipple and about half way between the left margin of the sternum and the vertical nipple line. It is systolic and often so small that only a careful observer would detect it. Moreover it is sometimes very variable in audibility, being scarcely noticeable with some beats and easily heard with others. Its characteristic feature is a twanging sound very like that made by twanging a piece of tin or string. This bruit is found mostly between the age of two and six years as a rule they are brought for some ailment such as a cough or some indigestion and the bruit is discovered only in the course of routine examination. It persists sometimes for many months. I have noted it as present in one case for two years. Whatever may be its origin I think it is clearly functional that is to say not due to any organic disease either congenital or acquired.

William Stokes (1801-1876) of Dublin (see p. 11) made many clinical observations on cardio-

vascular sound. In his textbook *Diseases of the Heart and Lungs* (1833) Stokes commented on two cases with "extensive and irregular" effusion of the aortic orifice and murmurs audible at a distance— at least three feet in the case of one. He noted that the very loud sound might be accompanied by little cardiac embolism.

The perception of these sounds was the principal cause of his suffering for his general health long continued excellent and the heart action was but little excited. This gentleman once observed to me that his entire body was one humming top. The humming was audible over the extremities in the case which was apparently one of calcific aortic stenosis.

William Sidney Thayer (1864-1932) of Baltimore (Fig. 5) made early studies of the physical third heart sound (1464) which with Hirschfelder (690) he discovered about the same time at the University of Edinburgh and about two years after Obrastzow of Kiev. He was impressed with the accentuated third sound of mitral regurgitation (1464). He pointed out that the third heart sound was loudest immediately after the subject assumed the recumbent position— so called *primo-decubitus* position (1468). Thayer thought the third sound was due to the sudden tensing of the mitral and perhaps at times the tricuspid valves at the time of the first and most rapid phase of diastole (1464). He introduced the term opening snap to correspond to the element d'ouverture of Ronchus (1467). He was early (1911) to describe an epigastric venous hum with hepatic cirrhosis (1466). With the pathologist William George MacCallum Thayer made studies of experimental aortic regurgitation in dog (1471).

Walter Hayle Halsey (1812-1891) after C. J. B. Williams, Professor of Medicine at University College Hospital made many observations on cardiovascular sound. He noted the change in quality of aortic murmurs on transition to the apex (1462).

Charles J. B. Williams (1807-1889) succeeded Elliot on a Professor of Medicine at University College. Williams invented the anamniopoeitic lubber top (1463). He appears to have described aortic sound in a case of complete heart block (1463). In studies sponsored by the British

Association for the Advancement of Science he devised the method of relating valve action to heart sound by putting his finger into the atrium of animal via the auricular appendage (1462). Of recent years the technique has been commonly used in mitral valve surgery.

MITRAL STENOSIS (67-462-1469) Writing in 1930 Cilio (221) made the following observation. Because of the multiplicity of its nomenclature, significance of the unit and character of certain of the and finally because of its own frequency, mitral stenosis probably is the cardiopathy which has motivated the largest number of clinical and phonocardiographic studies. For the reasons the history of the condition will be separately traced.

As we have seen earlier (p. 6) Corrigan may have described the diastolic murmur and thrill of mitral stenosis. Lacaze was probably familiar with both to some extent. In his 1826 edition he described the case of Louis Ponard, a 16-year-old gardener whom he had seen in 1822. The contraction of the aortic valve exceedingly prolonged took place with a dull bruit, strong and quite like the sound produced by a silk rubbing on wood. This bruit was accompanied by a purring sound which was heard by the ear and was evidently the same as felt by the hand. At the end of the contraction one heard a loud bruit accompanying the impulse and synchronous with the pulse. Since he thought the second sound represented the contraction of the atrium the prolongation of which he speaks was undoubtedly the diastolic rumble of mitral stenosis ending in a loud mitral first sound.

In 1824 J. R. Berthol (1767-1828) of Paris wrote a follow-up in his text on cardiac disease (93).

49th observation. Bellow murmur during the contraction of the aortic. Fibrocartilaginous degeneration of the mitral valve and mitral stenosis—A 68-year-old woman was seen in 1822 with a totally irregular pulse. The beats of the ventricle are irregular and intermittent; these intermittences in general are preceded by two quick rapid contractions which are closely

\* This reference appears to contradict the statement of Corrigan (204) that it is a more looked upon as unrelated phenomena.

He expounded the important concept that gallop sounds are in exaggeration of normal elements 'If one auscultates a goodly number of healthy persons, one will not be long in discovering that diastole is not always absolutely silent, that in the part of the cardiac cycle where the normal sound responsible for the gallop is located, there is sometimes already, in vestigial form, something which, when exaggerated, could be come the sound in question' Dock (197, p. 46) wrote as follows:

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\* Translation by Mounsey (1122)



speed. The contractions of the left ventricle have a very forcible impulse and rather a ringing sound, those of the auricles are accompanied by a murmur comparable to the blowing of a bellows, or even more to the placental murmur, the hand applied to the precordial region senses a vibratory motion deep but very marked; it is abruptly replaced by the ventricular movement.

It is clear that what Bertin heard was an unusually prolonged 'passive' diastolic murmur of mitral stenosis. In stating that it occurred with auricular systole he was following the authority of Lennec that the second heart sound is related to atrial systole. This view was not challenged until a few years later. Furthermore the total irregularity of the heart beat indicates that atrial fibrillation was probably present and a true auricular systolic murmur impossible. He also described the snapping first sound, the diastolic thrill and possibly the opening snap following the second sound (two quick rapid contractions).

The presystolic murmur was described and named by Sulpice Antoine Laue (1813-1884) *chef de clinique* at Hotel Dieu Paris in 1843 (43). I conclude from the facts stated in this memoir that a morbid presystolic bruit localized at the apex of the heart is in the existing state of science the stethoscopic sign which points with the greatest probability to contraction of the mitral orifice. In a footnote referred to the term presystolic he stated further: "I borrow the expression from M. Gendin (340) while admitting, that he used it in a different sense." A year later became one of the greatest of modern clinicians. He did work in public health in Turkey for 19 years and was later General Inspector of Health Services of France (44). This was a sharp departure from his clinical contributions as a young resident physician (fig. 7).

At approximately this same time Stoda pointed to enfeebled aortic sounds and accentuated pulmonary second sound (Skoda's sign) as an important clue to the diagnosis of mitral stenosis and Bouillaud related his *bruit de rappel* to this lesion. But most including Bouillaud, Laue's chief at the Hotel Dieu refused to accept Laue's description of the presystolic murmur.

In 1861 William Lennant Gardner (1824-1907) then of Edinburgh, later Regius Professor

of Medicine in Glasgow (341), also described the presystolic murmur and suggested the designation "auricular systole" (306). At about the same time Duroziez (392) invented his onomatopoeic device "font-tation" and gave lucid descriptions of "pure" mitral stenosis which resulted in the condition being known as Duroziez's disease in the French literature.

Finally, not long after 1871, when the convincing paper of C. Hilton Lagg (1848-1893) of Guy's Hospital, London, appeared (48), the presystolic murmur was accepted as the leading auscultatory sign of mitral stenosis. Lagg (48) quotes Hyde Siler (1823-1871), also of Guy's as stating in a student's lecture in 1869 that now anyone who should fail to recognize and identify this sound (presystolic murmur) would not only be unfit to hold the place of an accomplished and critical physician but could hardly be considered as a decently informed member of our profession.

Although the descriptive aspect of the presystolic murmur and its relation with mitral stenosis were well established a controversy long continued as to the genesis of the murmur. Gardner's auricular systolic concept was not accepted by all. An alternative interpretation which has been defended to the very present (see p. 293) was that the murmur is actually in early systole and is produced by regurgitation at the mitral valve.

Gardner (50a, 342) reported on a case illuminating in connection with the origin of the presystolic murmur. In 1861 he first saw an Irish labourer Patrick M. (about 20). He suffered no very great amount of inconvenience from his disease except from a remarkable undulating movement in his neck for which he came over to Edinburgh from Dundee about two years ago to consult Mr. Syme supposing that it was something that might be cured by surgery. Gardner found that the pulsation was venous and that there was an extraordinarily loud precordial murmur which resulted in the patient being long a show-case. The cardiac murmur begins immediately after the second sound, continues *diminuendo* throughout the pause and then goes on *crescendo* up to the first sound at which it stops abruptly. Gardner





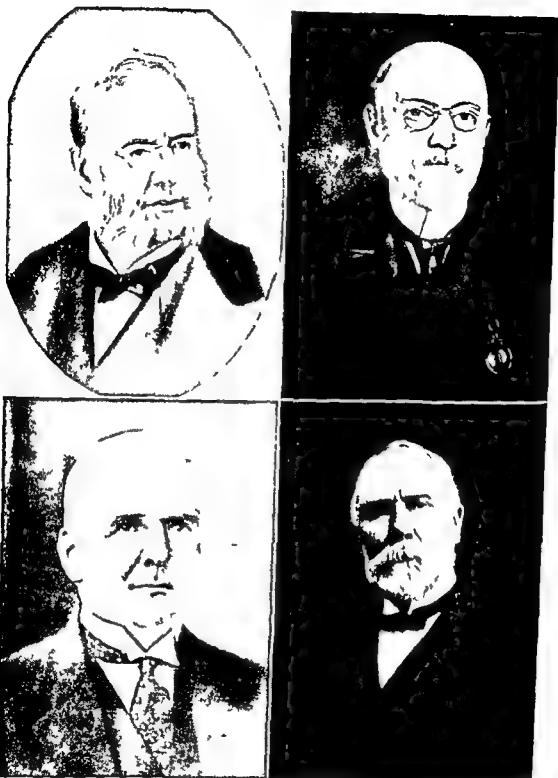


FIG 10 (Upper left) Austin Flint (upper right) Graham Steell (Courtesy of Dr A Morgan Jones, Manchester) (lower left) Carey Coombs (Courtesy of Dr John Cohen, Bristol) (lower right) James Mackenzie

the back, pursued them into the neck and even into the thigh (663). And he said of himself 'As long as my heart beats, I shall listen to the heart of others' (1868).

Among the many auscultatory signs which

Duroziez described is the waxing and waning of the diastolic murmur of tricuspid stenosis with inspiration and expiration respectively (405).

Graves used (or pointed) the usual area of maximal audibility of the murmur of aortic regurgitation

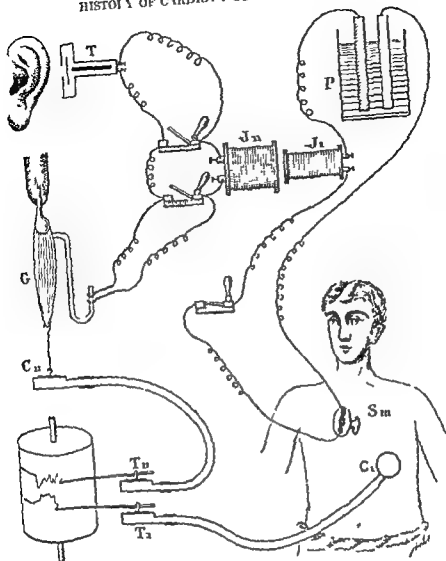


FIG. 12 Huerthle's apparatus using frog nerve-muscle preparation for registering heart sounds (1893)

ductorium the secondary coil of which was connected to the nerve of a frog nerve-muscle preparation which marked on a smoked drum by means of an attached lever (Fig. 12).

The first phonocardiograms in anything approaching the modern sense were made by Willem Einthoven (with Geluk) in Leyden in 1891 (418) using Lippman's capillary electrometer (Fig. 13)—with which the first electrocardiograms had also been made.

Direct phonocardiograms (Fig. 14) that is recordings of the precordial vibrations with optical amplification were first made (478) by Otto Frank (1855-1914) of Munich in 1904

(1533). This method making use of the so-called Frank segment capsule, was modified and improved on by many workers among them Carl J. Wiggers (1915-1918, 1921, 1922) in Cleveland, Ohio, and Orta and Braun Menendez in Argentina (1166). Using the direct method O. Weiss wrote an early (1909) monograph on phonocardiography in which many phenomena—e.g., arteriovenous fistula, splitting of heart sounds, stenosis and regurgitation at the aortic and mitral valves—were displayed graphically for the first time and the term 'phonocardiogram' used. The method suffered from limitations in the frequency response of the membrane used in the

Hospital, Boston, described (881) a scratchy systolic murmur in a high proportion of cases of severe thyrotoxicosis. Often the superficial and scratchy character is so pronounced as to suggest a pericardial friction rub. This is the so-called *German Means scratch* which is thought to be caused by high flow, in terms of both velocity and volume, through the pulmonary artery. As usual, earlier descriptions of the phenomenon can be found, for example in 1920 Goodall (567) wrote as follows: "A superficial pericardial rub is often heard, this is most common over the pulmonary base. It is probably produced mechanically."

In 1879 *Henri Roger* (1811-1891) of Paris described (1304) the long, systolic murmur of uncomplicated interventricular septal defect, the *Roger murmur*. In 1841 *Barth and Roger* had first published a textbook on cultivation, which went through many editions.

*Sjoda's* sign in mitral stenosis is referred to on p. 22 and p. 24.

In an article entitled 'the murmur of high pressure in the pulmonary artery' *Graham Steell*<sup>14</sup> (1851-1942) of Manchester, England (Fig. 10) described the murmur which bears his name (1436). Although this pulmonary diastolic murmur which is often difficult to differentiate from aortic diastolic murmur is usually associated in our thinking with mitral stenosis and probably indeed occurs most often in this situation it can as indicated by Steell occur with any type of pulmonary hypertension.

It will be noted for the admission among the recognized cultivatory signs of disease of a murmur due to pulmonary regurgitation such regurgitation occurring independently of disease or deformity of the valves and as the result of long continued excess of blood pressure in the pulmonary artery.

In cases of mitral obstruction there is occasionally heard over the pulmonary artery (the sternal extremity of the third left costal cartilage) and below this region for the distance of an inch or two along the left border of the sternum and rarely over the lowest part of the

bone itself a soft blowing diastolic murmur immediately following or, more exactly, running off from the accentuated second sound, while the usual indications of aortic regurgitation, afforded by the pulse, etc., are absent. The maximum intensity of the murmur may be regarded as situated at the sternal end of the third and fourth intercostal spaces. When the second sound is reduplicated the murmur proceeds from its latter part. That such a murmur as I have described does exist there in, I think, he has no doubt. The murmur of high pressure in the pulmonary artery is not peculiar to mitral stenosis although it is most commonly met with as a consequence of this lesion. Any long continued obstruction in the pulmonary circulation may produce it. The pulmonary valves like the aortic, do not readily become incompetent, apart from true structural changes. Probably no amount of blood pressure in the pulmonary artery will render them so suddenly as at least theoretically the mitral valves may be rendered incompetent. Changes in the vessel with widening of its channel and eventually, of its orifice long precede the occurrence of incompetence of its valves. The pulmonary murmur of high pressure is probably never persistent at first and one of its most remarkable features is as a rule, its variability in intensity. On some days it may be distinctly heard on others it will be indistinct or even inaudible while extreme accentuation of the pulmonary second sound is always present. The closure of the pulmonary semilunar valves being generally perceptible to the hand placed over the pulmonary area is a sharp thrill. An accentuated second sound is no less perceptible with a certain amount of incompetence of the semilunar valve on the contrary an accentuated second sound is associated with a regurgitant murmur is clinically common.

In 1894 *Pawlovsky* (1192) presented necropsy evidence in support of *Graham Steell's* view. It is probable that the *Graham Steell* murmur was described by *Hope* in 1812 (p. 18) what had been known for some time as *Hope's* early diastolic murmur was probably the same as what we now call the *Graham Steell* murmur.

*Indrag Traube* (1818-1876) of Berlin described (1474) a double sound heard over peripheral arteries in aortic regurgitation without pressure with the stethoscope. He found his sign also in cases of typhoid fever. He also made early studies of what he called *Galloprrhythmus* (1474).

#### THE ART OF PHONOCARDIOGRAPHY

An early (1893) method for making the heart sounds in eric their own record was that used by *K. Huchthle* of Breslau (720-721). He led the output of the microphone through an in-

<sup>14</sup> Steell (151) was the son of Sir John Steell the sculptor who designed the familiar monument to Sir Walter Scott in Princes Street, Edinburgh. He was a close friend of Mackenzie (see p. 20) who while in practice in Burnley near Manchester frequently made hospital rounds with him.

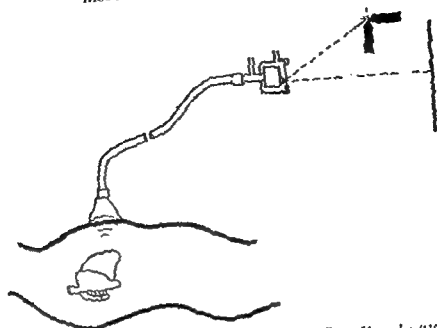


Fig. 14 Schema of direct phonocardiography (From Otis and Braun Menendez (1966))

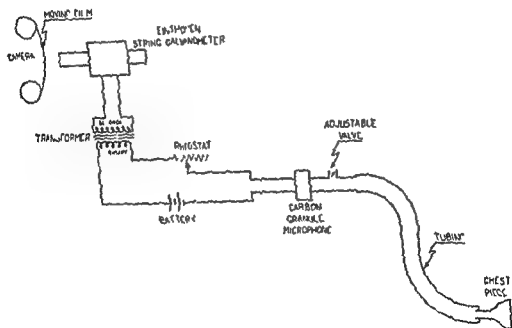


Fig. 15 Schema of Einthoven's string galvanometer phonocardiograph (124)

now a generation of *Heart Sound Indicators* totally unfamiliar with the string galvanometer put as the preceding generation was unfamiliar with the capillary electrometer.

In direct phonocardiography and in phonocardiography using the string galvanometer

since no amplifier was used and no electrical means for filtration or selective amplification were available it was necessary to use acoustical filtration to get rid of the low frequency vibrations of very large excursion which obscure the much faster vibrations in the audibility range of

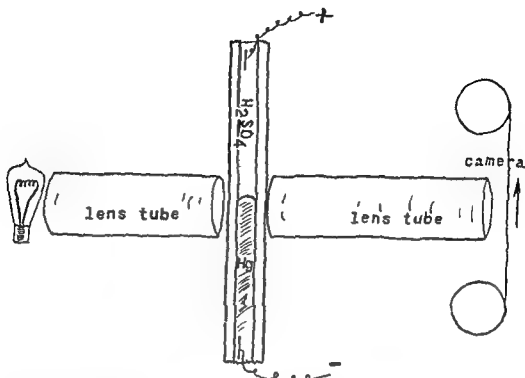


FIG 131 Schematic of Lippmann capillary electrometer (Adapted from Fig 202 p 643 W M Bayliss *Principles of General Physiology* London Longmans Green 1915 (70) )

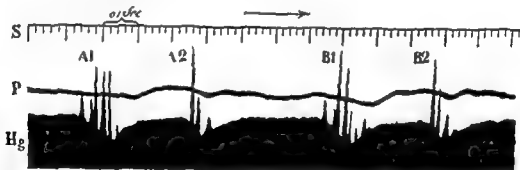


FIG 132 Phonocardiograms made with capillary electrometer (From Einthoven and Clarke (1894) )

segment capsule. Many of the phonocardiograms made with this direct method have an appearance suggesting that the undamped vibrations of the recording membrane were excited in a non-specific manner. In one publication (146) in connection with diastolic sounds demonstrated by this method it was stated, "since the ribs, intercostal spaces and sternum and indeed the whole body receive an impact during atrial systole and during rapid ventricular filling, the possibility is not eliminated that the eardrums might have set their highly undamped membranes into oscillation."

In 1907 (415) Einthoven<sup>16</sup> (1860-1927) who

<sup>16</sup>For an excellent photograph of Einthoven with his string galvanometer see the frontispiece of Burch and Wilson's *Primer of Electrocardiography*

in 1924 was awarded the Nobel prize principally for his work in electrocardiography reported on the use of the string galvanometer for recording the heart sounds (Fig 15). Although this method was used by Sir Thomas Lewis (902-904) by Bitterd (61) a pupil of Einthoven and others (311, 312) including in recent times Dock and Rydman the tremendous range of intensities encountered in cardiovascular sound far exceeded anything in the electrocardiogram led to frequent accidents with fracture or other damage to the string. Nonetheless much valuable information was uncovered. It is startling to consider that with the development of electronic amplification making possible use of various galvanometers such as the d'Arsonval and the advent of the cathode ray oscilloscope there is

the inverse relation ship between the duration of the interval between the second heart sound and the mitral opening snap and grade of mitral obstruction. These two features have been used in the last few years in the quantitative assessment of mitral stenosis (1a2a) (778).

The appearance of several monographs have been landmark in the history of phonocardiography and of cardiovascular sound in general.

1 Ortiz O and Braun Menendez C. [From Buenos Aires.] *The Heart Sounds in Normal and Pathological Conditions*. London: Oxford University Press, 1939.

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3 Weber A. [From Breslau.] *Herzschallregistrierung*. Dresden: Leipzig, Theodor Steinkopff, 1944.

4 Levine S A and Harvey W P. [From Boston.] *Clinical Auscultation of the Heart*. Philadelphia: W B Saunders Co, 1949.

5 Calo A. [From Tunis.] *Les bruits du coeur et des vaisseaux*. Paris: Masson, 1949.

6 Schmitt Volz J. *Herzdiagnostik in Klinik und Praxis*. Stuttgart: Georg Thieme, 1951.

Höllbach K and Wolf D. [From Heidelberg.] *Klinische und physikalische Lehrbuch der Phonokardiographie*. Stuttgart: Georg Thieme, 1956.

7 Weber A. *Atlas der Phonokardiographie Optische und magnetische Wiederanschauung des Herzschralls*. Jülich: Verlag der Herzschallregistrierung (1953). Darmstadt: Dietrich Steinkopff, 1956.

In addition Mannheimer of Stockholm produced a monograph length opus on calibrated phonocardiography which was published as a supplement to *Acta paediatrica Scandinavica* (1931). Butterworth of New York City (204) and Dixon of Denver (1244) have published monographs on cardiac auscultation and used phonocardiogram for illustrative purposes.

#### THE STRUCTURE AND FUNCTION OF THE HEART VALVES IN HEALTH AND DISEASE

So intimately is cardiovascular sound related to the heart valves that some waves of the development of knowledge about the anatomy, physiology and pathology of these structures is indicated. However, so extensive is this subject that only a brief review is possible.

STRUCTURE. According to Cullen (the original manuscript lost) Erasistratus who lived about

250 years B.C., was familiar with the heart valve and named the atrioventricular valves *tricuspid* and the arterial valves *sigmoid* (309). The early Greek letter sigma unlike the modern one was shaped like our letter C. The cup like shape of the cusps of the arterial valves is well indicated by the designation.

An unknown Hippocratic writer in *Pers. Kardies* described both sets of heart valves and the chordae tendineae (732). The origin of this brief piece is not clear although the majority of the Hippocratic writings are older it is possible that *Pers. Kardies* was written after Erasistratus.

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Culio Cesare Cranzio (Julius Caesar Crantius) of Bologna Italy (1540-1599) in his *Human Fetus* (1535) described the small cartilaginous nodule in the center of each semilunar cusp (31). These nodules are now called corpora arantii or the semilunar nodules.

Antonio Maria Valisavi (1660-1723) of Italy described the bulbus dilatation at the base of the aorta and pulmonary artery (1484). Valisavi's maneuver useful in investigating cardiac function would probably go by the name of some later discoverer had the observation on which it is based not been put on record by his student Morgagni (340).<sup>18</sup> If the glottis be closed after a deep inspiration and a strenuous and prolonged expiratory effort be then made such pressure can be exerted upon the heart and intrathoracic

<sup>18</sup> The quotation above is provided by Dixon (340). I have been unable to locate it in the translation of Benjamin Alexander published in 1799.

frequency. Acoustical filtration was accomplished by means of an air leak in the tube connecting the chest piece with the Frink capsule or microphone.

The advent of the vacuum tube and electronic amplification produced a change in the technique of phonocardiography in essentially all parts of the world. The review of Rappaport and Sprague (1244) in 1942 marked the end of any other methods of phonocardiography and outlined the use of electronic amplification and galvanometers of various types. Maurice B. Rappaport, Howard B. Sprague and their collaborators, of Boston have emphasized the separate areas of usefulness of what they term stethoscopic and logarithmic phonocardiograms (p. 79).

All phonocardiography hitherto discussed is oscillographic phonocardiography. A major departure was represented by adaptation of the Bell Telephone Laboratories' method of sound spectrography to the study of heart sounds and murmurs, by Geckeler and colleagues (532) who called it cardiospectrography, and by my colleagues and me (1085) who called the method *spectral phonocardiography*. Spectral phonocardiography is a development of this decade.

The following is a partial enumeration of some of the contributions made by phonocardiographic means or with phonocardiographic confirmation.

*Inthoven* (416) published the first recordings of the physiologic third heart sound.

In 1914 *C. J. Benjamins* (84), an ear nose and throat doctor in Utrecht demonstrated that an atrial sound can be recorded from the esophagus in all cases of sinus rhythm. In the same year *Fleeth W. Bridgman* (1888-1938) of Baltimore, published recordings of the atrial heart sound in normal boys (175).

In 1915 *Sir Thomas Lewis* (1881-1945) of London provided phonocardiographic confirmation for Mackenzie's observation that the plesystolic murmur of mitral stenosis disappears with the advent of atrial fibrillation (904). He provided one of the first published recordings of a musical aortic diastolic murmur of retroverted aortic cusps (902, Fig. 18).

In 1915 *Israel A. Wilson* (1890-1952), later of Ann Arbor, with Jameson presented satisfactory phonocardiograms of three cases of musical aortic diastolic murmur (1570). He noted

the decrescendo or crescendo decrescendo pattern of intensity. He noted that there was also a decrescendo in frequency. In his three cases, the frequency at the beginning was estimated at 150, 140, and 170 cps, and at the end at 125, 110, and 140, respectively.

In 1915 *Battaerd*, student of *Inthoven*, recorded atrial heart sounds in atrial flutter with heart block (61). He also studied extrasystoles.

*Charles C. Wolferth* (1887- ) of Philadelphia, working for the most part with *Alexander Margolis* (1894- ), reported studies which did much to familiarize the English-speaking world with the mitral opening snap (1040), pointed out the relationship of the intensity of the first heart sound to the PR interval of the electrocardiogram (1578), introduced the now generally accepted concept of summation gallop (1577). In 1935 from an analysis of the heart sounds in bundle branch block (1576), Wolferth concluded that the then generally held view that the right bundle was blocked in cases with the electrocardiographic pattern of so-called common bundle branch block, was false and that in fact there was block of the left bundle—an unusual triumph for phonocardiography.

*Camille Lian* (1882- ) of Paris, one of the leading phonocardiographers, has with various collaborators made many contributions to the precise description of cardiovascular sound. *Pleuropericardial systolic clicks* (920), the murmur of patent ductus arteriosus (935), the early diastolic snap of pericardial constriction (914), the early systolic click of disease of the aorta or pulmonary artery (939), musical extracardiac murmurs (913).

In 1937 in a beautiful article based on fourteen patients *Routier* (1316) demonstrated the pattern of the continuous murmur in patent ductus arteriosus.

In 1911 *Weiss and Joachim* (1522) pointed out that in mitral stenosis the first heart sound is delayed in relation to the QRS complex of the electrocardiogram and is compared with the situation in normal subjects. The phenomenon was rediscovered by *Cossio and Berconsky* (300) in Argentina in 1943 and by *Howard B. Sprague* and collaborators (1097) in Boston in 1941. *Sprague* and co-workers (1097) also pointed out

the inverse relation ship between the duration of the interval between the second heart sound and the mitral opening snap and grade of mitral obstruction. The e two features have been used in the last few years in the quantitative assessment of mitral stenosis (1523) (778).

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7 Hollnack K and Wolf D [From Heidelberg] *Atlas und Lehrbuch des Lehrbuch der Phonokardiographie* Stuttgart Georg Thieme 1950

8 Weber A *Atlas der Phonokardiographie* Opische und magnetische Aufzeichnung des Herzschalls zugleich 2 Auflage der Herzschallregistrierung (cf 3) Darmstadt Dietrich Steinkopff 1950

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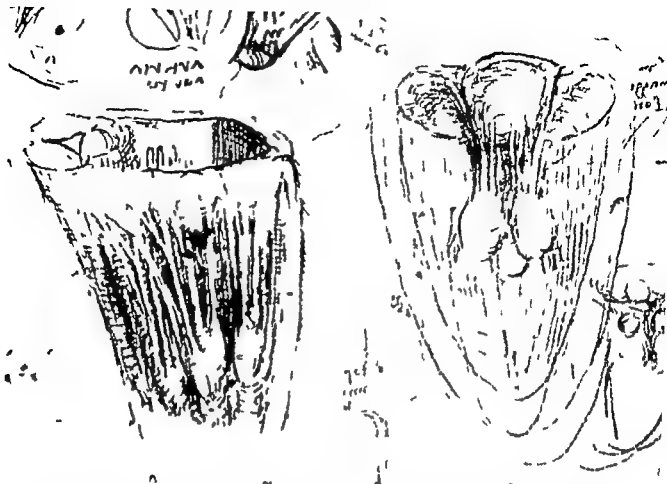


FIG. 16 Drawings of the functional anatomy of the heart by Leonardo da Vinci. A (left) Tricuspid valve open. B (right) Tricuspid valve closed. (From O. Milnes and Saunders (1158).)

vessels that the movement and flow of the blood are temporarily arrested.

In 1840 Karschner (829) described small myocardial fibers in the atrioventricular valves. These fibers are basically atrial in origin and have been suggested as a contributing factor in valve closure, a dubious theory.

Discoid valves, particularly mitral and aortic, were described by John Mayow (1613-1670) of Oxford (1034). Théophile Bonet (1620-1689) of Geneva. Vicussens (1641-1713) of France. Ingersi (1654-1720) of Italy (1594). Albrecht von Haller (1708-1777) of Switzerland. Jean Baptiste Senac (1693-1770) of France. Giovanni Morgagni (1682-1771) of Padua. Matthew Baillie (1761-1823) and William Cowper (1666-1709) of England (313) and others before Lennet.

**FUNCTION.** The functional anatomy of the heart valves fascinated Leonardo da Vinci (1452-1519) who left notes and drawings (Fig. 16) which are now the property of the British royal family and

are housed in the library at Windsor Castle (1158). In general he had a clear notion of the anatomy of the valves and portrayed their structure in a much clearer manner than did, for example, Vesalius illustrator. Clearly, specifically, at least two functional observations were made. (1) By analysis of the geometry, Leonardo established that a three-cusp aortic valve provides maximal strength in the closed position and minimal obstruction to forward flow when open. (In 1936 in a clinicopathologic report Kissin (798) demonstrated that a quadricuspid pulmonary valve is prone to regurgitation.) Furthermore, a bicuspid valve produces mild obstruction to forward flow such that a murmur may be produced and atherosclerosis and bacterial endocarditis develop secondary to the locally altered pattern of flow. In 1873 Longworth<sup>17</sup> (963) Pro-

<sup>17</sup> Longworth's reasoning was somewhat different. He concluded that in order to open (without obstruction to flow) the sum of the lengths of the free borders of the cusps should equal the circumference of the aortic

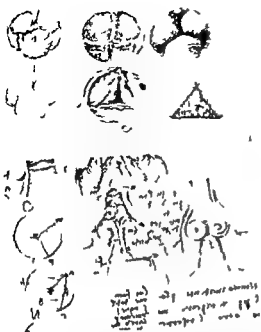


FIG 16 C (above) Semilunar valve open and closed (several views). Note triangular shape of orifice when valve is open. (2) Below: Vortex formation behind cusps and hypothetical role of vortices in closure of semilunar valves. (From O'Malley and Saunders (115b).)

Professor of Anatomy in the Medical College of Ohio, Cincinnati came independently to the same conclusion as Leonardo about the superiority of a tricuspid artificial valve. (2) Leonardo conceived of vortices behind the semilunar cusps being responsible for a partially closed position of the cusps during ventricular ejection and for subsequent closure of the valve. The same idea—that eddies behind the valves open up like tightly wound watch springs and close the valve when ventricular ejection ceases—was expressed by Krehl in 1891. The existence of vortices of the type drawn by Leonardo in the sinuses of Valvula during ventricular ejection is theoretically likely on hydrodynamic ground. Such vortices may be important in the formation of the murmur of Valvula in illustration of the role of hemodynamic factors in the morphogenesis of the

organ and the length of the free borders of one cusp should equal the diameter. By means of a model of paper he illustrated to his class that a tricuspid valve most readily satisfies the requirement. The lengths of the free borders of the three cusps approximate the circumference of the circle  $\pi \approx 3.1416$  (value

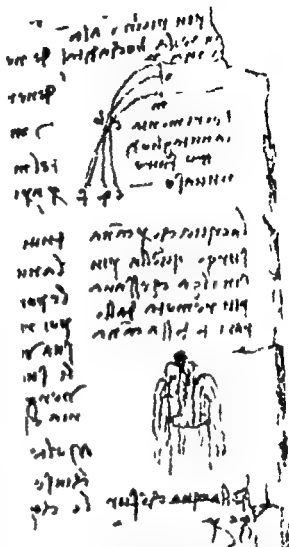


FIG 16 E Leonardo visualized the formation of vortices by a method analogous to the way the water falls in flowing from a horizontal or upright tube. (From O'Malley and Saunders (115b).)

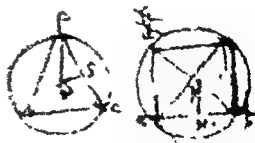


FIG 16 F Drawing to indicate that the quadracus valve is weaker because of greater height of the triangle (as drawn here by Leonardo). (From O'Malley and Saunders (115b).)

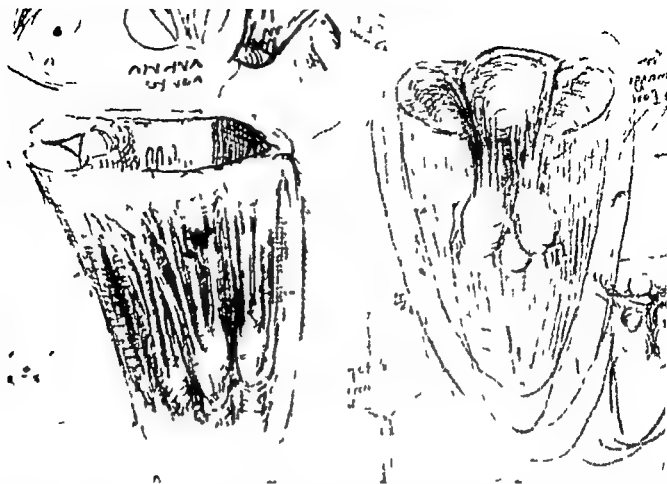


FIG. 16 Drawings of the functional anatomy of the heart by Leonardo da Vinci. A (left) Tricuspid valve open. B (right) Tricuspid valve closed. (From O. Mallory and Saunders. (1958).)

vessels that the movement and flow of the blood are temporarily arrested.

In 1840 Kuschner (529) described small myocardial fibers in the atrioventricular valves. These fibers are basically atrial in origin and have been suggested as a contributing factor in valve closure—a dubious theory.

Diseased valves, particularly mitral and aortic, were described by John Mayow (1643–1670) of Oxford (1034), Théophile Bonet (1620–1689) of Geneva, Vicussens (1641–1715) of France, Janesio (1654–1720) of Italy (1094), Albrecht von Haller (1708–1777) of Switzerland, Jean Baptiste Senne (1693–1770) of France, Giovanni Morgagni (1682–1771) of Padua, Matthew Baillie (1761–1823) and William Cowper (1666–1709) of England (313), and others before Lennec.

**FUNCTION.** The functional anatomy of the heart valves fascinated Leonardo da Vinci (1452–1519) who left notes and drawings (Fig. 16) which are now the property of the British royal family and

are housed in the library at Windsor Castle (1108). In general he had a clear notion of the anatomy of the valves and portrayed their structure in a much clearer manner than did, for example, Vesalius, the illustrious Celsus. Specifically, at least two functional observations were made: (1) By analysis of the geometry, Leonardo established that a three-cuspid aortic valve provides maximal strength in the closed position and minimal obstruction to forward flow when open. (In 1936 in a clinicopathologic report Kustin (796) demonstrated that a quadricuspid pulmonary valve is prone to regurgitation.) Furthermore, a bicuspid valve produces mild obstruction to forward flow such that a murmur may be produced and atherosclerosis and bacterial endocarditis develop secondary to the locally altered pattern of flow. In 1873 Longworth<sup>17</sup> (963) Pro-

<sup>17</sup> Longworth's reasoning was somewhat different. He concluded that in order to open (without obstruction to flow) the sum of the length of the free borders of the cusps should equal the circumference of the aortic

Carwell (1793-1857) (240) Marc d'Espine (1037) Carwell came to the conclusion from examining on the service of Louis in Paris a patient with an aneurysm of the ascending aorta presenting to the right of the sternum. Over the body of the heart the first and second sounds were faint (making impact of the heart against the chest wall unlikely as a mechanism) but over the tumor the second sound was maximal and occurred at the systolic swelling of the tumor (140 p 151). But none established the origin of the second sound more convincingly than did Joseph Rouanet (1797-1865) of Paris in his thesis for the M.D. degree (1832). He affixed a bladder in the outflow tract of the ventricle in such a manner that he could simulate ventricular systole by "squeezing it. Then listening over the arterial valve he heard a sound like the second heart sound when he relaxed pressure on the bladder (Fig 17 1).

Billing (103) had attributed both heart sound to tension of valve curtains, the atrioventricular valves in the case of the first sound the arterial valve in the case of the second. But it was almost exclusively intuition that led him to this conclusion. On the other hand Rouanet (1314) who likewise held that both sound were due to tension of the valves as much as or more than the actual collision of the cup margin provided experimental evidence for this view. Rouanet stated at the beginning of his thesis that he would cover the following topic:

(1) I will briefly review what has been published on the subject (2) I will show that action of the heart valves is accompanied by sound (3) that these sounds coincide with those of the heart and that in granting that these are the same one provides perfect explanation for the observed fact (4) that no existing theory is admissible (a) I shall end with certain considerations of the orifices and valves of the heart and abnormal sound.

Rouanet's experiments consisted of (1) the use of the model (Fig 17 1) described in the text below in which arterial valve action was simulated (2) the use of a second type of model in which action of both the mitral and the aortic valve was simulated and (3) the tensioning of membranes and relating of the character of the sound produced to the physical structure of the

membrane. (The second type of experiment was described in his second publication in 1844 (417).)

I put the part below the sigmoid valves around a glass tube about an inch in diameter and 2 or 3 inches long joining below a bladder similarly fixed to it and full of water. The portion of the vessel above the valve was fixed to the lower end of a second tube of the same diameter and more than four feet in height in order to compensate by the elevation of the liquid column for the forces of impulsion which normally exist between it on the part of the blood or of the arteries and the neighboring part. Then raising the apparatus at the level of the valves which remain free and applying my ear in such a manner that it is separated only by the finger, I would squeeze the bladder suddenly with the other hand I would imitate insofar as possible the beating of the heart as to the quantity of liquid which I made pass into the upper tube with each beat and as to the intermittence of the action which I exert

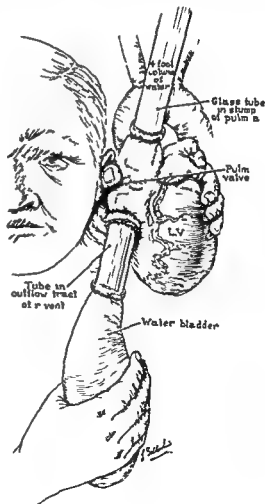


FIG 17 1 Rouanet's experiment (see text)

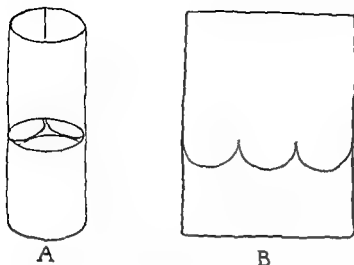


FIG. 17 Longworth's model (see text)

cardiovascular system. The partially closed or at least mid position of the valves during ventricular ejection is now well established. Although it is of interest how far the genius of Leonardo was able to penetrate into the mysteries of valve function, historically his studies were of no significance because they were completely unknown until relatively recent times.

It has probably been evident since Harvey at least that the closure and opening of valves is fundamentally the result of a change in polarity of the pressure differential on the two sides of the valve. The details of the mechanism of valve closure were filled in later however. The ancillary or contributory mechanisms account particularly for the fact that the valves close without attendant regurgitation when the mechanisms go wrong, regurgitation is likely to develop.

In the instance of the semilunar valves Ceradini (248) established in 1873 that the valve assumes an intermediate position of partial closure during ventricular ejection. He suggested that at the end of ejection the cusps are pulled together in the wake of the systolic ejection jet and that the margins of the cusps are virtually in contact at the time that the polarity of pressure differential is reversed.

A similar mechanism in the closure of the atrioventricular valves was supported by the work of Luciani about 1903, Yandell Henderon (670) with Johnson about 1912, and Dean about 1916 (341). Henderon in particular emphasized the importance of atrial systole in assisting closure of the atrioventricular valves, by a breaking of the

jet" mechanism comparable to that in the closure of the arterial valves (cf p 112).

Beginning with Roumet, who reported in 1832 (1314), and Baumgarten in 1843 (63), various authors have built systems to simulate the pumping action of the heart and study valve action. An interesting example is the system used by Grid and illustrated in his publication (502) in 1886. In recent years Ian McMillan (1088, 1089) of London adopted this method, combined with cinematography, for the study of directed valves in human beings and others (331, 803) have followed suit. Cinematographic studies of heart valves have been performed (1107) in excised, surviving, and perfused hearts of animal. In intact animals Rushmer had made very cinematographic investigations of the movement of heart valves—to which radiopaque markers have been affixed (1326).

#### THE PHYSICS AND PHYSIOLOGY OF CARDIOVASCULAR SOUND

**THE HEART SOUNDS.** The error of Lennec's view that the first and second sounds are due to ventricular and atrial systole respectively was first pointed out in 1829 by John William Turner (1790-1836) Professor of Surgery (?) in Edinburgh who had only to recall that Senac, Lincis-Hiller and a number of others knew that the atrium contracts immediately before the ventricle, not after the ventricle (1477). One of his students described Turner as a timid shy man who could not look his class in the face and seemed fitted by nature for anything rather than the duties and responsibilities of an operating surgeon (588). Perhaps he was particularly fitted by nature for challenging Lennec's doctrine! On his premature death Turner was succeeded by the famous Sir Charles Bell.

Although other theories continued to be embraced by some workers for several decades (see below) shortly after 1830 the second sound was related to closure of the arterial valves by several writers among them Billing,<sup>18</sup> Robert

<sup>18</sup> The prize of the century for comprehensive titles is certainly due Billing's *On the auscultation and treatment of the affection of the heart*—all in an article three and one half pages in length!

and in tantancon force we are obliged to agree that it would produce a sound perceptible to the ear

Rouanet related the difference in character of the two heart sounds to physical difference in the heart valves. The first sound is loud and depends to a certain extent on the energy of the ventricle and is duller than the second. The valve which occasions it are larger and the walls which conduct it thicker. The second sound is harper because the valves are smaller thinner and attached to more anorous walls.

Rouanet of some special interest to American cardiologists is because he spent the last eight years of his life as a practitioner in New Orleans. Born in southern France in 1797 the son of an illiterate peasant Pournet (213-344-447-1075) was befriended by the proprietor of the estate where his father worked. He was educated for the priesthood being sent to seminary in Paris about 1820. However he soon renounced his ecclesiastical intentions and undertook to earn a livelihood by teaching Latin and Greek. His formal medical education was between 1828 and 1832. From the time of qualification until 1847 he practiced medicine in Paris. Because of failure to enlarge the circle of his clientele and also because of heavy losses in stock market speculation he emigrated to New Orleans in 1847. There he appears to have been an important addition to the medical community. An entertaining epilogue is described by a colleague and friend (447).

One day a young colleague wished to have a joke at Rouanet's expense. He brought to Rouanet's house very gravely an anatomical specimen saying it was an infant's heart which showed most singular anomalies. It seems that Rouanet studied them with much curiosity. But it was the heart of a goose! I do not know to what extent Rouanet was deceived by it. What is certain is that he took it very poorly. This impropriety or more this thoughtless news had wounded him on a sensitive point. Another colleague made the mistake of recounting the episode in a bit of doggerel verse which circulated about town. This was too much! A duel with pistols ensued. Happily after the first shot which grazed both of them the witnesses serious-minded colleagues declared honor satisfied. Rouanet wished to begin again.

Further details on the episode of the goose heart and the ensuing duel is provided by Tinker (1073). It was Rouanet's long time rival Dr

Charles Chaurin Bonclair Déléry (1815-1880) who composed and circulated the poem entitled *Le médecin et l'oie*.

As examples of other views on the origin of the heart sound held by our trading contemporaries of Rouanet three may be cited. James Hope in the first edition of his textbook (1831) attributed the second sound to an impact occasioned with attainment of full diastolic filling of the ventricle. The great François Magendie (1783-1855) believed (1022) that both major sounds are due to impact of the heart against the rib cage the first sound with ventricular contraction the second sound with ventricular filling. The evidence he assembled for this (each bit obviously has an alternative explanation which escaped his attention) was as follows. Attenuation of the heart sound when air and fluid surround the heart seeming correlation between the strength of the heart beat and the intensity of the heart sounds, difficulty of hearing any sounds from the exposed heart except those which to his mind could be attributed to impact of the heart on the monaural stethoscope. In regard to Rouanet's theory of the heart sounds Magendie stated as follows. This explanation is physically inadmissible it supposes the existence of a void in the ventricle and the two great arterial trunks. It is probably true that Rouanet performed his experiments tensing membranes in air. Magendie apparently questioned whether a ripple would result when the tensing was performed in liquid such as blood. Dock (357) has repeated Rouanet's experiments in liquid.

The controversy over the origin of the first sound continued unabated despite Rouanet's convincing demonstration. Over a century later a leading cardiologist was still writing as follows (153). It is generally believed that the principal factor in production of the first heart sound is the sudden increase in the tension of the muscle fibres of the ventricle.

In England a considerable amount of investigation on the origin of the heart sounds was undertaken in the late 1820's and the 1830's. Every where indicated by the titles of the period the motions and the sounds of the heart were investigated simultaneously. It had early become evident that to understand both heart sounds

ced on the bladder. At the moment when my fingers which had just squeezed the bladder abruptly relaxed to let the liquid descend, a very marked shock would strike my ear it was audible as often as I repeated compression of the bladder. Its force was in relation to the height of the fluid column. It was very analogous to the second sound of the heart. I saw analogous and not very similar such no one would expect to find a perfect resemblance here, since the conditions are so different the pulmonary artery has been submitted to the same experiment with the same results.

He described the second type of model in the following words:

The apparatus I most often use is composed of two reservoirs communicating with the heart by tubes. The lower reservoir designed to furnish the liquid, is raised 3 or 4 centimeters above the organ. It tube connects with the left auricle. The second tube 2 to 2½ meters long connects the aorta to the higher reservoir of which the capacity like that of the first is 8 to 10 liters. At the apex of the heart is fitted a third very short tube which carries below a caoutchouc bulb. If one opens the tap of the lower reservoir, the water enters the heart and rises on the side of the aorta after having filled the rubber bulb and ventricle.

To produce valve action it is only necessary to imitate systole and diastole by sudden compression and release of the rubber bulb. The first of these two movements causes the bicuspid *claquement* to be heard the second the semilunar *claquement*.

From the results of his experiments he made important deductions about pathological alterations in the heart sounds: the dull first sound in mitral regurgitation the loud second sound in arterial hypertension, the *tumbe* and *clac* of origin of murmurs and so on.

**FIRST SOUND.** Its force is related to the energy of the contractions of the ventricles and to the exact occlusion of the auriculo-ventricular orifice the which preceded with unusual weakness we should look into whether ventricular systole lacks vigor and whether the large valves close their orifices incompletely. In the first case the movements of the heart have small excursion and the apex beats slightly in the second the heart seems to move forcibly but the pulse remains very compressible and if the ear perceives a murmur it is heard most distinctly toward the apex of the heart and not toward the place corresponding to the origin of the arteries. Such was undoubtedly the malady of Marguerite Johoval (45th observation in the work of Messieurs Bertin and Boulaud) "Beats of the heart soft but of large excursion

pulses scarcely palpable on the two sides. The heart is very voluminous the mitral valves are ossified." All these phenomena are very well explained by the return of blood into the cavity of the auricle.

The first sound which is normally dull can become even more so by thickening and by passage to a cartilaginous or osseous state.

**SECOND SOUND.** Its force depends (1) on the fullness of the arterial vessels which therefore react with more energy on the vessel and push it back more slowly against the sigmoid valves. (2) On the rapidity of ventricular contractions. It passes in a given time a greater quantity of blood across the widely opened arterial orifice the valves, finding them elastically widely spread, thereby give with the retrocession of the column of blood more excursion than they would if they were only feebly opened. (3) On the complete occlusion of the orifice by the sigmoid valves if there remains between them a gap which permits blood to reflux toward the heart the shock is considerably diminished. The first cause is evidenced by easy compressibility of the arteries the second by the little energy of the heart and the slowness of its contractions measured by the first silence [systole]. The third should cause an abnormal sound to be heard during diastole of the heart which corresponds to the second silence.

In regard to timbre the second sound displays considerable changes when the sigmoid valves acquire an abnormal consistency for example whenever they are cartilaginous osseous or simply thickened the superior [second] sound naturally clear will acquire a very striking peculiarity which permits prediction of one of the alterations.

Let us hasten to add that the shock in the sense of what our ordinary ears and which result from the collision of two bodies is not the only cause of the valve sound. Rather numerous experiments have shown me that all membrane in passing suddenly from fluidity to tension always produces a sound which varies according to the circumstances. Its force is in proportion to the thickness of the membrane. Its *éclat* merges with the thickness and inextensibility of the tissue which composes it. The size, thickness and extensibility of the membrane render the sound more dull. The body to which it is attached also influences considerably the quality of the sound by its thickness, softness and elasticity. In accordance with their texture and action the auriculo-ventricular valves combine the most favorable conditions for the production of the sound they are thin resistant inextensible they pass in an instant from the most complete fluidity to sudden and violent distension resulting from the impulse of the blood and the traction of the numerous tendons which pass from their margin and ventricular face to attach at the apex of several enormous columns. Consequently whether we consider in the valve a surface which goes to strike abruptly against another surface or whether we see in it an eminently *onctuous* membrane subjected to a strong

and in tantaneous force we are obliged to agree that it should produce a sound perceptible to the ear

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and murmurs it was necessary to have a clear view of the sequence of cardiac events. The nature and timing of the apex beat must, it was clear, be established. Although the finishing touches had to await the development of the recording kymograph by Carl Ludwig (1816-1895) about 1847 and of recording techniques by Chauveau, Murey and others, important points were established by experiments such as those of James Hope, famous also as a clinical cardiologist (see p. 18). The technique for one of Hope's experiments is described in this manner on August 21, 1830, "an ass, of which the pulse and impulse were forty eight per minute was instantaneously deprived of sensation and motion by a sharp blow on the head. The trachea was opened, a large bellows pipe introduced and artificial respiration maintained, while at the same time, the left ribs were sawn through near the sternum, and forcibly bent back and broken, so as widely and completely to expose the heart immediately behind the left shoulder the whole was accomplished in less than five minutes."

The British Association for the Advancement of Science sponsored studies of the basis of cardiovascular sound in several successive years in the 1830's. Investigations were made by Carlisle (1834) (230), Robert Adams<sup>19</sup> I. W. Green and others (1835) (8), C. J. B. Williams (see p. 23), Todd, Clendinning, and others (1836) McCurtney, Adams, Kennedy and others (also 1836), and Clendinning (1840) (273). Some of the conclusions were undoubtedly false. For example the committee of 1835 thought the first sound too long to be valvular in origin and concluded that it is due mainly to ejection of blood over the rough ventricular lining although the bruit musculure might contribute. The studies did further establish that the second sound is related to closure of the semilunar valves and that impact against the

thoracic cage plays no role in the genesis of the first and second sounds.

The famous *Jean Cruveilhier* (1791-1874), who held the first chair of pathology in the Paris faculty and whose name is memorialized in the Cruveilhier Baumgarten syndrome, communicated the findings in an infant with ectopneustic cords (318). He could hear almost no sound over the ventricle but heard both sounds over the great vessels. This led him to the notion that the first sound is produced by the flapping open of the arterial valves and the second by closing of these valves.

A muscular contribution to the first heart sound or even in origin of the first sound solely on this basis was long favored. In his Croonian lecture of 1809 (1083), William Hyde Wollaston (1766-1828) physician, physiologist, physicist and chemist, wrote on the sound produced by contracting skeletal muscle the muscular murmur" as he called it. He pointed out that by obliterating the external auditory meatus with the thumb and tensing the muscles of the arm one can hear this muscle sound thereby demonstrating that muscular effort which appears to be unitary is in fact a composite of contractions occurring at a frequency of 20 or 30 per second. Helmholtz thought it unlikely that the first sound arises predominantly in the myocardium since the sound produced by contracting muscle is usually low pitched.

Auguste Chauveau (1827-1917), veterinarian then in Lyons (Fig. 20) using horses did experiments relating the heart sounds to valve function (203). Chauveau was in the department of anatomy of the veterinary school. Operating on a meager budget he felt compelled to make fullest possible use of the decrepit horses available to him for dissection. He would perform physiologic experiments in the morning, before slaughtering the horse for dissection in the afternoon. In one famous series of experiments performed about 1845 he rendered the horse immobile by transfixing the cerebrospinal axis at the level of the medulla. Respiration was maintained by insufflating the trachea with a large bellows. It was a matter of popular interest that the animal in this preparation although incapable of voluntary motion could still sugar and evidence

<sup>19</sup> Robert Adams (1791-1875) surgeon to Queen Victoria and Regius Professor of Surgery at Dublin was an unusual surgeon. He wrote in 1827 about the condition to which his name along with Stokes is attached. He made important studies of mitral stenosis recognizing the thrill, the high incidence of arrhythmia and the pulse deficit. He reminds one of the contributions to the understanding of mitral stenosis made by surgeons in recent years.

appreciation for same Chauveau would open the thorax and introduce the hand into the left atrium via the auricular appendage. By this method he convinced himself that the first sound proceeded from the apex of the atrioventricular valve. In many of these experiments Chauveau had the collaboration of Jean Fèvre, a young physician who has the further distinction of having made the first direct intra-arterial measurements of arterial pressure in man (1830). He performed these studies in patients undergoing amputation of a limb and used Poiseuille's hemodynamometer—a mercury-filled L tube.

With the help of Marey (see p 47) Chauveau did the first cardiac catheterization in horse about 1860 using balloon catheters (Fig. 18) introduced through the jugular vein. The time of occurrence of the heart sound was indicated manually and correlation made between intra-cardiac events and the heart sound (Fig. 19). Visitors to the laboratory expressed amazement that the animal munched hay peacefully as the cardiac phenomena were being recorded (Fig. 20).

In 1859 Donder's in Holland recorded the apex beat in man and marked the timing of the heart sound by manual means. Thus he did in man what Chauveau had done in the horse. An important result of the experiments of Chauveau and Donder was the demonstration of the nature of the apex beat. Prior to that time it had been held by many that the apex beat related to atrial systole. Accurate identification was of considerable importance to the accurate timing of murmurs.

MURMURS. In Dublin Dominic John Corrigan (see p 17) did important early (1829) experiments producing murmurs by compressing arteries in living animals and by compressing length of intestine through which he caused water to flow (29a). The conclusions of these studies which were directed towards elucidation of thrills (frémissement caténaire cat purring) as well as murmurs (bruit de soufflet bellows sound) were stated in this manner by Corrigan:

- 1 That bruit de soufflet is not owing to pulsation
- 2 That frémissement caténaire and bruit de soufflet are identical
- 3 That they do not depend on any particular vital



FIG. 18 The prototype of the modern cardiac catheter (1855). The atrial and ventricular balloons are indicated by 'a' and 'v' respectively. One lumen of this double lumen catheter communicates with the atrial balloon and one with the ventricular balloon.



FIG. 19 A recording of intraventricular pressure (1860) with the time of the heart sound marked manually (Fèvre) (1855).



FIG. 20 This medallion was subscribed for by a group of Chauveau's friends including Burdon Sanderson of Oxford and presented on January 23, 1903. The medallion was designed by Dr. Paul Richer, physician, sculptor and friend of Chauveau. A brochure describing the presentation ceremony was distributed to the subscribers. *Alta naturae scrutatus mente vigenti* (meaning 'Scrutinizing the occulteries of nature with a lively mind') is an appropriate motto. It is equally appropriate that his most famous experiment is portrayed.

condition of the heart and artery but on a purely physical cause.

4 That that cause is an alteration in the motion of the blood instead of its equable progressive motion en masse it moving as a current producing impulses

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The famous Jean Cruveilhier (1791-1874), who held the first chair of pathology in the Paris faculty and whose name is memorialized in the Cruveilhier Braungarten syndrome, communicated the findings in an infant with ectopia cordis (318). He could hear almost no sound over the ventricle but heard both sounds over the great vessels. This led him to the notion that the first sound is produced by the flapping open of the internal valves and the second by closing of these valves.

A muscular contribution to the first heart sound or even in origin of the first sound solely on this basis was long favored. In his Croonian lecture of 1809 (1583) William Hyde Wollaston (1766-1828), physician, physiologist, physicist and chemist, wrote on the sound produced by contracting skeletal muscle, the muscular murmur as he called it. He pointed out that by obliterating the external auditory meatus with the thumb and tensing the muscles of the arm one can hear this muscle sound thereby demonstrating that muscular effort which appears to be unitary is in fact a composite of contractions occurring at a frequency of 20 or 30 per second. Helmholtz thought it unlikely that the first sound arises predominantly in the myocardium since the sound produced by contracting muscle is usually low pitched.

Luguste Chauveau (1827-1917) veterinarian then in Lyons (Fig. 20) using horses did experiments relating the heart sounds to valve function (263). Chauveau was in the department of anatomy of the veterinary school. Operating on a meager budget he felt compelled to make full use of the decrepit horse available to him for dissection. He would perform physiologic experiments in the morning before sacrificing the horse for dissection in the afternoon. In one famous series of experiments performed about 1855 he rendered the horse immobile by transfixing the cerebro-spinal axis at the level of the medulla. Respiration was maintained by insufflating the trachea with a large bellows. It was a matter of popular interest that the animal in this preparation although incapable of voluntary motion could sit up and evidence

<sup>19</sup> Robert Adams (1791-1875) surgeon to Queen Victoria and Regius Professor of Surgery at Dublin was an unusual surgeon. He wrote in 1827 about the condition to which his name along with Stokes is attached. He made important studies of mitral stenosis recognizing the thrill, the high incidence of arrhythmia and the pulse deficit. He reminds one of the contributions to the understanding of mitral stenosis made by surgeons in recent years.



upon the sides of the heart or arteries and recording sensations

He added

To produce *bruit de soufflet* it is not necessary, however, that there should be in every instance contraction of some part of the arterial tube, or unnatural dilatation, or when in the heart that there should be narrowing of one of the valvular communications *bruit de soufflet* accompanies simple dilatation of the ventricles. From what has been said it will be easily understood that the dilated ventricle and natural sized opening into it bear the same relation to one another as the natural sized ventricle and contracted opening.

All of this is indeed remarkable, considering that it was written only ten years after announcement of the stethoscope. Corrigan further pointed out that

*bruit de soufflet* is heard without any lesion either of contraction or dilatation with which to connect it—in hysterics, nervous irritable patients, in patients suffering from immediate evacuations, from hæmorrhage in the very weak and irritable.

In the last group Corrigan attributed the murmur to some more vague disturbance of blood flow. More precise elucidation had to wait description of the role of viscosity of the fluid and velocity of flow in determining the pattern of flow.

The second report of the London committee of the British Association (p. 42) with C. J. B. Williams as principal member addressed itself to the genesis of murmurs. Confirmation for Corrigan's observations and conclusions was provided by study of flow through croutchouze tubes. Grating, or rasping, sounds were best obtained by the action of a strong current on a lotted thread across the diameter of the tube. Some of the experiments were repeated with water rendered glutinous with size and it was found that the sounds were not so readily produced as with plain water and required a greater force of current. This explained the fact that murmurs occur more in the living body, in states of anaemia when the blood is thin and more like water.

Rouanet (p. 39) in his historic thesis on the origin of the heart sounds (1832) discussed murmurs also.

*Le bruit de soufflet* indicates the rubbing ("frottement") of the blood against the orifices of the heart. It has stenosis as its usual cause. It can also be produced when the ventricle enlarged in capacity or more rapid in its movements causes the blood to pass with much rapidity across an orifice which remains in its natural state. This is what occurs in a large number of cases in which the *bruit de soufflet* persists only a short time.

*Les bruits de scie* and *de raie* are only two variations on the same symptom. Their cause should be found in a sort of trembling ("tremblement") and vibration impressed on the column of blood by some body which oscillates with its pressure.

Can we now recognize which of the four orifices of the heart is the site of origin of the abnormal sound? Two circumstances serve to tell us: the time in the heart rhythm when it is heard and the area of the thorax where the ear perceives it most distinctly. If the abnormal sound corresponds to the first silence, or the contraction of the ventricles, its cause is found almost always at the arterial orifices, especially if it is a *bruit de raie* or *de scie*: its maximal intensity will be three inches above the cardiac apex and little to the right. However this same type of murmur can have its seat at an auriculo-ventricular orifice which is poorly closed by the valves; it is then toward the cardiac apex that the sound is distinguished with more force. What I have just said occurs in the inverse order when the abnormal sound is produced during diastole of the ventricles. Most often it is stenosis of the auriculo-ventricular orifices which causes it: the ear appreciates it best toward the cardiac apex. Nevertheless it can happen in certain circumstances that the sigmoid valves altered in texture permit the blood which has just reached the aorta to reflux toward the heart with production of various types of noises of which the maximum intensity is found at the same point of the chest as that of the physiologic second sound.

It will be noted that although it did not interfere with his physics, Rouanet's understanding of the physics of murmur production was imperfect. However, after twelve more years spent in the study of cardiovascular sound, Rouanet arrived at a more accurate theory of the cause of murmurs. He insisted in 1844 (447) that it was in the formation of eddies, vortices, or turbulence that their basis is to be found. The word he used *tourbillons* is noteworthy. It is almost superfluous to remark on the facility with which turbulence (*tourbillons*) and murmurs are produced by abnormal dilatation of the arteries, stenosis of orifices, the presence of blood clots, and the insufficiencies.



FIG 21 Photograph of a marble bust of Savart by Etax (1841). This is the only likeness of Savart known to be in existence. The bust is preserved at the Académie des Sciences in Paris.

In the 1830's in Paris Felix Savart (1791-1841) by diploma a physician but in actuality a physicist and principally an acoustician (Fig 21) did pioneer experiment on sound generation in liquid systems (13:2). Figure 22 is an artist's conception of two of the models with which Savart experimented. Water is permitted to flow from the reservoir through a small orifice at the bottom (B). A pure tone is generated by this set up through two interlocking forces—the hedding of vortices (or a Savart called them fluid vein) at the orifice and resonance in the fluid column. The rate or frequency of vortex hedding is determined by the height of the water in the reservoir. The resonating frequency of the water column is likewise a function of its height. As the water falls in the reservoir the frequency of vortex hedding falls. A sound produced in connection with the vortices at the orifice is

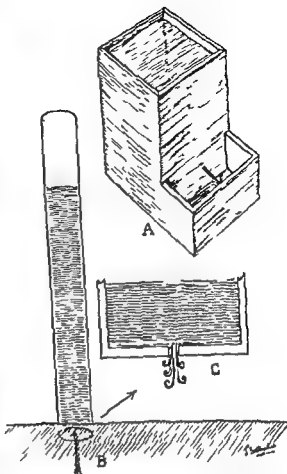


FIG 22 Two of the jet experiments of Felix Savart (author's concept from Savart's description). A and B. Overall view of the two models. C. Detail of orifice plate and vortex hedding in B. The approximate dimensions of the model shown in B were as follows: the (13:3) closed the lower end of a glass tube 6-8 cm in diameter and 1.2 m long with a metal plate pierced at the middle by a cylindrical opening whose diameter (was) equal to the thickness of the plate.

intensified to the level of audibility when the natural resonating frequency of the water column matches the frequency of vortex hedding. The net effect is that as the level of water falls in the reservoir a series of pure tones of successively lower pitch is produced each tone being excited from the next by a short period. The similarity of the model pictured in Figure 22B to the situation in vortex stenosis and the fact that Savart trained as a physician was in Paris during the exciting period immediately after Linnec, stimulate speculation as to whether Savart's experiments had a medical motivation. A side

from this interesting thought, the importance of his experiments lies in the demonstration that sound can be generated in flowing fluid. During the rest of the last century Savart's "fluid veins"—call them eddies, or vortices, or even turbulence, if you prefer—are encountered repeatedly in writings on the mechanism of murmurs. In some of his experiments Savart made the "fluid veins" visible by means of a dye, usually indigo, a method exploited by Reynolds 50 years later.

In 1841 Bouillaud wrote "The precise determination of the conditions under the influence of which the different variations in murmurs are produced constitutes one of the most perplexing problems at present and one of the most difficult in bioacoustics. It would indeed be desirable if one of our Savarts would apply himself to the solution of a problem which surely is not unworthy of the full attention of the most competent physicists." This would appear to indicate that Bouillaud was unaware of any medical implications of Savart's experiments. However, Savart died in 1841 and his experiments which are perhaps most relevant to cardiovascular sound were reported posthumously in 1864 (1352).

An earlier contribution of Savart has pertinence in connection with cardiovascular sound. In 1826 (1301) Savart studied the velocity of sound in liquids and observed that sound travels much less rapidly in liquid contained in a tube with elastic walls.

In the 1830's *Chamneau*, the veterinarian (p. 42), studied murmur production by essentially the same methods as did Corrigan—he compressed the pulmonary artery in the horse and also studied compressed tubes through which fluid flowed—and came to conclusions identical to Corrigan's. However, familiar with the work of Savart, he attributed the murmur, not to "an alteration in the motion of the blood" (Corrigan) but specifically to "fluid veins"—which amounts to the same.

In 1850 *Franz Alexander Knirsch Ritter von Rotterau* (1814–1852), versatile professor of obstetrics and gynecology in Würzburg reported (799) studies into the physics of cardiovascular sound and came to the following conclusions as reviewed by Skoda (1393). Knirsch had found murmurs at the dilatations in crutchhouse tubes.

"Water flowing out of a pipe preserves to a certain distance the form of the pipe's outlet, in this way the stream, flowing from the narrow into the wider part of the tube returns to a certain distance the form of the narrow part now, in consequence of the pressure of the air, the walls of the wider part of the tube have a tendency to adapt themselves to the narrow stream of water, but their elasticity offers a continual resistance to the pressure of the air. Under these conditions, the tube, alternately compressed by the atmosphere and expanded by its own elasticity, is made to vibrate, and by its vibrations a murmur is produced. In the last ten years Rodbird (p. 138) has espoused what is essentially the same mechanism.

Skoda quoted the following conclusions directly from Knirsch: "The first sound of the heart is produced by the expansion of the auriculo-ventricular valves, the second sound, by the expansion of the semilunar valves. Murmurs arising in consequence of a defect of the valves of the heart are produced in part by the vibration of the rigid valves, but more particularly after the manner described in the experiments with crutchhouse tubes. Pressure upon an artery causes a murmur just beyond the spot compressed, never at that spot. The so-called *lun's murmur*—*bruit de diable*—is invariably formed in the carotid artery and not in the cervical veins it is caused, in fact by the compression of the carotid by the omohyoideus muscle. Muscular, or any other kind of pressure, will in like manner produce this murmur in other arteries of persons whose blood is impoverished."

In 1805 (1517) studies of the genesis of murmurs in models were reported by *Theodor Weber* (1829–1899) of Leipzig later Professor of Medicine at Halle (1228). His father, Ernst Heinrich Weber (1793–1878) of Leipzig was one of the pair of brothers who singly or jointly discovered among other things (1) the cardio-inhibitory influence of the vagus nerve (1845) (2) the finite velocity of the arterial pulse wave disproving Bichat's view that the pulse is felt synchronously in all arteries (3) the relation of response to stimulation in sensation including hearing (the Weber-Fechner law). In many ways Theodor Weber's study on murmurs was com-



FIG. 21 Osborne Reynolds (1842-1912)

Reproduction from a painting by Sir John Collier which now hangs in the Department of Engineering of Manchester University. (Courtesy of Professor Robert Platt, Department of Medicine, Manchester.)

comparable to that of Bondi done about 80 years later (129).

Chauveau, collaborator Marey (1870-1904) in his notable *Le circulation du sang* (1881) described (1079) studies in a model imitating circulation. When the resistance of the model which corresponded to peripheral arteriolar resistance was low the murmur was less than when it was high. A paradoxical increase in flow with increase in peripheral resistance has recently been pointed out by Rodbard (1249) in a similar model. In connection with the venous hum Marey pointed out that the murmur was most intense in diastole of the ventricle at which time flow in the large vein in the neck is perhaps most rapid.

In the last quarter of the nineteenth century Osborne Reynolds (1842-1912), Professor of Engineering in Owens College, now the University of Man-

chester in England (11, 23), made physical experiments of the greatest importance to the understanding of murmur production. In two sets of experiments Reynolds demonstrated for the first time with such clarity, and analyzed in fair detail, the phenomena now known as turbulence and extinction. The experiments on turbulence were reported to the Royal Society in 1884 under the title "An experimental investigation of the circumstances which determine whether the motion of water shall be direct or sinuous, and of the law of resistance in parallel channels" (1261). By "direct" Reynolds meant what we would call laminar or streamline flow. By "sinuous" he meant what we would term turbulent flow. For these investigations he used the set up demonstrated in Figure 24 and rendered the character of flow visible by injection of a dye into the flowing stream of water. The model consisted of a large tank with glass walls filled with water. Inside the tank was a long straight glass tube at one end opening into the tank by a

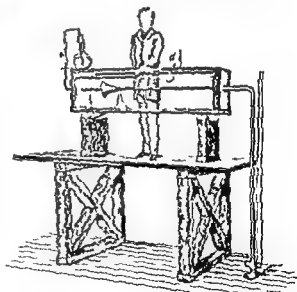


FIG. 24 The model built by Reynolds to demonstrate turbulent and streamline flow. A long glass pipe with attached funnel end drained off water from the tank when the valve was opened by means of the long handle. The small bottle sitting above the water tank contained dye which via the small glass tube down in the sketch could be injected into the stream of water leaving the tank. It is evident how experimental velocity could be investigated in such a model. From Reynolds' publication (1261).



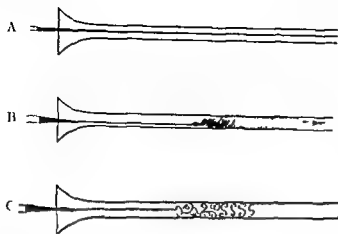


FIG. 25. Patterns of flow demonstrated by the model shown in Figure 24. *A*, Streamline flow at velocities below the critical level. *B*, Turbulent flow at velocities of flow in excess of the critical level. *C*, Semi-schematic illustration of vortices in turbulent flow as viewed by intense light. (From Reynolds' publication (126).)

funnel and at the other connecting to a drum. A smaller tube permitted injection of a dye into the center of the stream. Using the long handle of the model, Reynolds could vary the rate of flow in the tube by opening the valve to a variable extent. He could also vary the diameter of the pipe and the kinematic viscosity (absolute viscosity divided by density) of the liquid.

By this series of experiments, Reynolds demonstrated that at low rates of flow the dye maintained streamlines, but that above a certain critical velocity, flow became turbulent (Fig. 25). Furthermore, the greater the diameter of the pipe and the less the viscosity of the fluid, the greater was the tendency to turbulent flow. These considerations Reynolds incorporated in a formula for a value now known as the Reynolds number:

$$RN = \frac{\text{velocity of flow} \times \text{diameter of conduit}}{\text{kinematic viscosity of fluid}}$$

The Reynolds number is dimensionless. If the conditions of the basic model are fulfilled particularly as to the existence of a long, straight, smooth-walled tube, any liquid will undergo transition from streamline to turbulent flow when the parameters of flow are varied in such a way that the Reynolds number exceeds about 2000. The Reynolds formula is rarely applicable in anything approaching a quantitative way in the

cardiovascular system. Furthermore, it is not proper to equate turbulence and murmur directly, since, among other complexities, an interplay exists between the disturbed flow pattern and the wall (or protuberance) is important in murmur production. However, the Reynolds formula is a useful catalogue of factors influencing the development of murmurs in the cardiovascular system.

Cavitation was described by Reynolds in 1891 in a communication entitled, "The boiling of water at ordinary temperatures" (126). He showed that when water is caused to flow with sufficient velocity through a tube with a constriction in it (see Fig. 99), bubbles form at the constriction because of the marked drop in pressure at that point according to Bernoulli's principle. In essence, the water boils locally, as indicated by Reynolds' title. Reynolds thought the bubbles might be dissolved gases or the vapor phase of the liquid itself or a combination of the two. Beyond the constriction, the liquid flow forms a swirling stream cloudy with bubbles. At a point further downstream, the bubbles collapse, producing a hissing sound. The structural analogies between the Reynolds cavitation tube and certain lesions of the cardiovascular system, such as aortic stenosis and coarctation of the aorta, suggests a possible role of cavitation in the genesis of the murmurs associated with these lesions. To date, this remains only a possibility, however.

A method for approximate quantification of the intensity of the heart sounds and murmurs for clinical purposes was devised by Hermann Vierordt (1831-1932), son of Karl von Vierordt (1818-1881) and reported in 1883 (149). Vierordt had a cylindrical monaural stethoscope, roughly the original Laennec stethoscope with multiple segments. He would add segments to the stethoscope until the sound being studied was no longer audible. The length of the stethoscope at the point of disappearance of the sound in question was taken as a measure of its intensity.

Vierordt later made important contributions to the understanding of congenital heart disease (1490). Still later he wrote a book on the illnesses of famous people and the role of such illness in shaping history (1493). His textbook of percussion

and auscultation first published in 1884, had gone through 22 editions by 1930 (1492)

### THE VOCABULARY OF CARDIOVASCULAR SOUND

*Stethoscope* was a term suggested by Laennec. It is derived from Greek words meaning chest and to look at.

*Auscultation* is a word much older than *stethoscopy*. In comparing the sound of the heart to the sound of a horse's galloping, Harvey wrote *qui auditum fuit et pulum quindm et in cultantibus et tangentibus exhibit* (see p. 3 for translation). Brown (187) says that Forster was the one who applied the term auscultation to Hippocrates' famous method of examining for hydro-pneumothorax. Bui on (1776-1802) of Paris wrote in 1802: "There are two kinds of audition, one which is passive and involuntary and continuously practiced in waking hours, and a second which is active and produced by the influence of the will on the ear. The term audition is itself appropriate for the first type. I shall designate the second by that of *auscultation*. I define auscultation then as the exercise of the will in audition."

I pointed out earlier (p. 8) Laennec referred to the first and second heart sound as *bruit ventriculaire* and *bruit auriculaire* respectively. They were also referred to at times as the *systolic* and *diastolic* sound or the *inferior* and *superior* sound. By 1850 or earlier the current usage became well established.

It is difficult to trace the origin of the designations *aortic*, *pulmonic*, *tricuspid* and *mitral* for the major areas of auscultation (p. 72).

The French use of the word *bruit* for the heart sound is appropriate since they are in fact noises. The German equivalent *Herzton* some time later literally translated into English as *heart tones* and into French as *tons du coeur* is inappropriate because of the accepted nonmedical practice of reserving the term *tone* for vibrations of pure frequency, i.e., of one frequency or with a small number of harmonics in integer ratio. The French use the term *bruit* also in the generic sense to indicate both murmurs and heart sounds. Note for example the title of Lido's monograph: *Les bruits du coeur et des vaisseaux*. The generic

designation used here *cardiovascular sound* has the same significance.

In general, regurgitation, insufficiency and incompetence have been used interchangeably. The latter two are less precise than the first. In describing the aortic valve lesion in 1419 wrote "An ob-sticulum ne quid penitus regurgitaret effluere frunt imponere." (then quoted by Harvey) had used the term *reflux* and Harvey followed suit. Cowper (313) in describing a diseased aortic valve in 1780 used the verb *regurgitate*. Allen Burns used it in his textbook of pathology (1879).

*Incompetence* is used particularly by British writers. *Insufficiency* by American authors. Some authors tend to use *incompetence* for regurgitation at an arterial valve, *insufficiency* for regurgitation at an atrioventricular valve. Another quirk is to reserve *incompetence* for what some authors refer to as *relative* insufficiency and to use *insufficiency* for regurgitation due to organic lesion of a heart valve. The New York Heart Association approves of the word *insufficiency* and *incompetence* and makes a distinction between them. On page 76 of its publication (1979) the following statement is made: "Valvular incompetence is due to dilatation of the valve ring, it is to be distinguished from valvular deformity with insufficiency in that there is no structural alteration in the valve leaflet." The title of a recent article (1317) seems ridiculous at first thought but can be seen to be in the best tradition of the New York Heart Association nomenclature. Confusion of tricuspid incompetence with mitral insufficiency.

In *insufficiency* and *incompetence* are objectionable because they are non-precise terms even a stenotic valve is insufficient and incompetent in the performance of one valve function to wit opening, without objection to forward flow.

*Inadequacy* would be as good a word as term for insufficiency or incompetence. In fact Corrigan (213) writing in 1832 stated: "I have been in the habit for some years of describing this disease under the name of inadequacy of the aortic valves." He explained that I shot-on in his elegantly written work *Diagnosis of the Heart*.

For it would be impossible to imagine that the aortic would permit regurgitation downwards.

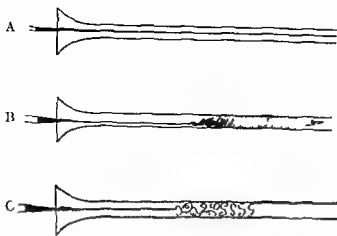


FIG 2a Patterns of flow demonstrated by the model shown in Figure 24 A Streamline flow at velocities below the critical level B Turbulent flow at velocities of flow in excess of the critical level C Semi schematic illustration of vortices in turbulent flow as viewed by intense light (From Reynolds publication (1265) )

funnel and at the other connecting to a drum. A smaller tube permitted injection of a dye into the center of the stream. Using the long handle of the model, Reynolds could vary the rate of flow in the tube by opening the valve to a variable extent. He could also vary the diameter of the pipe and the kinematic viscosity (absolute viscosity divided by density) of the liquid.

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"For it would be impossible to imagine that the obstacle would permit regurgitation downward!

suggested the designation *permanent patency of the aortic valve* which Corrigan used in the title of his article. In another part of the article he used the term "inefficiency." In 1861 Duroziez (393) spoke of "aortic insufficiency" in describing his double arterial murmur—Duroziez's sign.

The best practice, in my opinion, is to use *regurgitation* exclusively, since this more precise term leaves no doubt of the physiologic situation present. Admittedly "relative regurgitation" would be awkward, if not absurd. "Non cuspid" or "non valvular regurgitation" might be suitable, or as Wildman (1000) suggests, "secondary regurgitation." Also it might be argued that "insufficiency" and "incompetence" refer to an attribute of the valve, namely, its lack of water tightness whereas "regurgitation" is the functional consequence of the attribute.<sup>1</sup> However, since the attribute *per se* is of no significance and only its consequence is physiologically and clinically of note, *regurgitation* remains, in my view, the term of choice.

*Murmur* is derived from the Latin word with the same meaning. It is obviously onomatopoeic. Occasionally in the past the word *susurrus* borrowed directly from the Latin and meaning a "hissing," was used to mean murmur (e.g. 1343). *Bruit* and *souffle* are vestiges of the early days when no attempt was made to translate terms borrowed directly from French e.g. 'rattle' *bruit de souffle* for 'bellows sound' (blowing murmur) *bruit de scie* for 'sawing sound' (a variety of musical murmur), *fremissement caire* (cat purr) for 'thrill' etc.

*Reduplication*, *duplication*, and *doubling* of a heart sound were used in the past for splitting but are objectionable because true reduplication has not occurred—only components normally present become evident. Use of the term might have been justified in an earlier period when systolic clicks and gallops and other sounds in diastole, such as the opening snap and the

<sup>1</sup> The minor difficulties encountered when one always uses *regurgitation* is illustrated by my statement on p. 5 that Douglas found at autopsy aortic regurgitation. It was of course an incompetent valve not regurgitation he found.

## L'Assiette au Beurre [ LES MÉDECINS. ]



FIG. 26 a and b Two cartoons caricaturing use of direct (immediate) auscultation (or at the most the monaural stethoscope) by French physicians. a Cover of *L'Assiette au Beurre* for March 22, 1902 (No. 51). Title of issue *Les medecins*. Artist Abel Faivre. b Caricature of direct auscultation. I. Ange gardien. Artist Hoffman. (Donated by Dr. Lawrence Freeman, New Haven.)

periodical constriction snap had not been fully described and distinguished from true splitting.

In the last few years the terms *pansystolic* and *holosystolic* have been used interchangeably to refer to a murmur which extend through the entirety of systole. Since both prefix use of



Fig. 2 and 3 Two more cartoon caricatures of direct auscultation and the monaural stethoscope. From Weber & Talleu de la caricature médicale Paris 1936 d'après l'original de l'artiste. (Lentil by Dr. Moore)

(Greek origin) is to be there is no objection to either of these terms. However, *holo-systolic* is probably preferable since it can mean only what is indicated above, whereas *pan-systolic* carries the meaning of each. A *pan-systolic* murmur might equally well be one occurring in diastole. Although the latter meaning is usually false it is not what the users of one or the other of the terms have in mind.

The term *gallop* dates from Bouillaud who according to Potain (1224) was using it at least as early as 1847. Evans (412) has suggested the more general use of the designation *triple rhythm* as much as the distinction of gallops from their physiologic counterparts is often difficult. Other (p. 942) in 1911 on the desirability of maintaining the term *gallop*.

Some older writers used the term *diminuendo* in lieu of *decrecendo*. They are probably synonymous. The Oxford dictionary states: "Further more the 1911 edition of Grant's Dictionary of Music and Musicians comments as follows: 'Whether there was originally any difference between *decrecendo* and *diminuendo* or not at present the two terms appear to be convertible'.

Batterard a student of Linnthoven used the term *cardiophonogram* (61). *Stethocardiogram* has enjoyed some use in the past (1339). In 1910 (22) Jewell & F. Barker (1867-1943) of Baltimore, and in 1911 Kahn (764) used the term *phonocardiogram* which is now preferred and almost universally used. Barrington Bone and Lockhart (102) and later Dunn (788) favored the term *electro-stethograph*.

### THE REGISTER SIGN

As referred to earlier direct auscultation was practiced by the majority of physicians in France in preference to stethoscopy. The cartoons reproduced in Figure 26 derive from this fact.

Someone has proposed a system of grading of murmurs which from the most subtle and sophisticated at one end of the scale down to the most obvious and blatant at the other goes as follows: Cardiologist's murmur, physician's murmur, surgeon's murmur, administrator's murmur.

In 1848 Dr. Oliver Wendell Holmes (see p. 12) wrote an amusing poem about a young stethoscopist who took his newly acquired tool too seriously.



You use your ears all you that can  
But don't forget to guard your eyes  
Or you may be cheated like this young man  
By a couple of silly abnormal flies

### EPILOGUE

In the last century excessive emphasis may have been placed on the findings of auscultation. Preoccupation with cardiovascular sound probably directed attention away from important varieties of heart disease particularly coronary artery disease. James B. Herrick (1861-1934) who was important in pointing out the clinical features of acute coronary occlusion recognized the emphasis on physical diagnosis as a factor in the delayed recognition of coronary artery disease (672). Ernst von Leyden (1832-1910) an early student of coronary artery disease wrote (907) that in his century most physicians seemed to think that a heart in which no auscultation and percussion could reveal nothing abnormal must be healthy and conversely a heart in which one could hear anything unusual must be seriously diseased. This despite the warnings of physiologists such as Latham (p. 19) and of William Stokes of Dublin (see p. 22) who Herrick later (673) warned against over-estimating physical signs and who said that vital physical signs should be still regarded as of value as they had been in the pre-auscultatory period.

Mackenzie did considerable service in emphasizing the importance of the state of the myocardium in determining the clinical course of heart disease. However he may have underestimated the significance of valvular heart lesions and the auscultatory signs associated therewith. In the English speaking world cardiovascular sound no longer enjoyed in the first half of this century the intense interest which surrounded it in the last century. Phonocardiography never attained quite full respectability as an investigative technique.

Successful surgery of valve lesions has brought a considerable revival of interest in cardiovascular sound. Auscultatory details which previously would have been considered mere minutiae have now become in some instances literally matter of life or death. One can examine no better case in point than the so-called opening snap of mitral stenosis. Boullaud 120 years

ago was probably referring to it among other conditions in his *bruit de rappel* and Duroziez in 1861 in the second of his four treatises. It was beautifully described and assigned its present designation by Rouches seventy years ago. In spite of this almost no new textbooks of cardiology or of physical diagnosis written in English in this century mention it. The paper of Margolies and Wolferth in 1933 appears to have occasioned little interest and attention. Most textbooks and clinicians persisted in teaching that the double sound heard in mitral stenosis is a split pulmonary second sound. Since 1918 when surgery for mitral stenosis was revived by Charles I. Bailey of Philadelphia followed by many others the opening snap has once again been rediscovered and assigned an important role in distinguishing predominant stenosis from predominant regurgitation and in assessing the grade of mitral obstruction and the result of operation.

Mackenzie mentioned that mitral regurgitation is a benign lesion. He quoted and heartily subscribed to the statement of Crutcher Steell that 'no one ever dies of mitral incompetence'. This view held was in Anglo-Saxon cardiology for the first half of this century. Again the possibility of urgent treatment of mitral stenosis made it clear that mitral regurgitation is a significant lesion if for no other reason than that when combined in any significant proportion with mitral stenosis the results of surgery are less than optimal.

What is likely to be the course of the study of cardiovascular sound in the future? Will the changing pattern of heart disease and/or the introduction of new diagnostic techniques and refinements of the old reduce the importance of auscultation? Will the stethoscope be replaced by a more refined instrument or will vital diagnosis of the sound be relied on more to the exclusion of the ear?

Since two major varieties of heart disease—rheumatic and syphilitic—can be expected to become relatively rare in the rather near future the usefulness of the stethoscope will decrease greatly. However gallop will continue to be important as a signal of a laboring myo-



# THE STETHOSCOPI SONG A PROLIFERATION BALLAD

by

Oliver Wendell Holmes

There was a young man in Boston town,  
He bought him a stethoscope nice and new,  
All mounted and finished and polished down,  
With an ivory cup and a stopper too.

It happened a spider within did crawl  
And spun him a web of ample size  
Wherein there chanced one day to fall  
A couple of very imprudent flies.

The first was a bottle fly big and blue  
The second was smaller, and thin and long,  
So there was a concert between the two  
Like an octave flute and a tawny song.

Now being from Paris but recently  
This fine young man would show his skill  
And so they gave him his hand to try  
A hospital patient extremely ill.

Some said his liver was short of bile  
And some that his heart was oversize  
While some kept arguing all the while  
He was crimmied with tubercles up to his eyes.

This fine young man then tubercled he  
And all the doctors made a pause  
Said he: The man must die, you see  
By the fifty-seventh of Louis's laws.

But since the case is a desperate one  
To explore his chest it may be well  
For if he should die and it were not done  
You know the autopsy would not tell.

Then out his stethoscope he took  
And on it placed his curious ear  
Mon Dieu! said he with a knowing look  
Why here is a sound that's mighty queer!

The bourdonnement is very clear —  
Amphoric buzzing, as I'm alive!  
Five doctors took their turn to hear  
Amphoric buzzing and all the five.

There's emphysema beyond a doubt  
We'll plunge a trocar in his side  
The diagnosis was made out —  
They tapped the patient so he died.

Now such is late new-fashioned toys  
Began to look extremely glum  
They said that rattles were made for boys  
And vowed that his buzzing was all a hum.

There was an old lady had long been sick,  
And what was the matter none did know  
Her pulse was slow, though her tongue was quick  
To her this knowing youth must go.

So then the nice old lady sat,  
With phials and boxes all in a row,  
She asked the young doctor what he was at  
To thump her and tumble her ruffles so.

Now when the stethoscope came out  
The flies began to buzz and whizz  
Oh ho! the matter is clear no doubt  
An incurium there plainly is.

The *bruit de rixes* and the *bruit de seen*  
And the *bruit de diable* are all combined  
How happy Bouillaud would be  
If he could see like this could find!

Now when the neighboring doctor found  
A case so rare had been described  
They every day her ribs did pound  
In squads of twenty so she died.

Then six young dunces slight and frail  
Received this kind young doctor's care  
They all were getting slim and pale  
And hoarse of breath in mounting stair.

They all made rhymes with sighs and ke  
And loathed their puddings and buttered roll  
And dined, much to their friends' surprise  
On pickles and pencils and chalk and coal.

So fast their little hearts did bound  
The frightened insects buzzed the more  
So over all their chests he found  
The rick-siffant and the rick-somere.

He shook his head: There's grave disease —  
I greatly fear you all must die  
A light post mortem if you would please  
Surviving friends would gratefully.

The six young dunces wept aloud  
Which so prevailed on six young men  
That each his home-t love avowed  
Whereat they all got well again.

This poor young man was all right  
The price of stethoscopes came down  
And so he was reduced to clear his throat  
To practice in a country town.

The doctors being very sore  
A stethoscope they did devise  
That had a hammer to clear the bore  
With a knob at the end to kill the flies.

## CHAPTER 2

# Notes on the History of Respiratory Sound

Reference to respiratory sound are much more clearly identifiable in the ancient literature than are references to cardiovascular sound. The Simon uccusio n s p l i h of Hippocrate i i c i s e in point. Pleural friction rub and rales were probably known and direct auscultation was presumably practiced in their detection. The Simon p i s s i g e in Hippocrate compari n g the sound to those accompanying the boiling of vinegar u g r e s t bubbling rale.

An early reference to the Hippocratic uccusio n (1170) i s continued in a letter written in 1679 by Sir Thomas Browne who described a woman or maid in Suffolk who had a yulking and fluctuation in her chest so that when he stood and stroked her chest it might be heard by the bystanders by and I once heard it. He died as I remember about a veire and half after and in her chest was found a ves ti contain n g about a quart i I take it of a matter like thick whive of the Dr Furber now of Woodbridge gave an account to the B S (Royal Society) about even veire past and it is printed. See references 4 B.

Despite its longer history auscultation has in the case of respiratory sound not fared as well as it has in connection with cardiovascular sound. Where a much information unobtainable by other clinical method is provided by auscultation of the heart a r a y although it has certainly not replaced pulmonary auscultation completely i h i made it less essential. Physical exploration of the chest will remain in the hands of the expert a valuable supplement to the radiologic exploration. A combination of the two can provide a more complete evaluation than either alone.

In a recent examination sent respiratory disease was the only sign of leukochole carcinoma. X rays and all other tests were normal.

The larger share of Lacunec's work was in the field of respiratory sound. He described the phenomena of pectoriloquy and egophony and assigned these names to them. In a 28 year-old woman Lacunec found in listening below the right clavicle that her voice seemed to issue directly from the chest and to pass unaltered through the central canal of the instrument. He observed the same phenomenon which he termed pectoriloquy in 20 other patients all of whom had cavities discovered at autopsy. Egophony he described as a high pitched sharp silvery, quavering, jerking sound like the bleat of a goat—and of course the term means goat sound. He stated: Its occurrence appears to me to be restricted to subject suffering from acute and chronic pleurisy with a limited amount of effusion in the pleural cavity.

Rale meaning rattle was used by Lacunec in his writing but in his ward rounds the Latin *rhonchus* was substituted because rale connoted death rattle to the French patient of the day. He classified rales into (1) moist rales or crepitation (2) mucous rales or gurglings (3) dry sonorous rale or roaring and (4) dry subant rale or whistling. He stated that dry sonorous rales not infrequently resemble the cooing of a dove. This resemblance is often so close that one is tempted to believe that a dove is hidden under the bed. This statement is similar to his comment about the musical venous hum (which he incorrectly supposed to rise in the arteries of the neck) that one might at first think it issued from a musical instrument being played in the apartment below.

Lawson Brown (1871-1937) physician at the Trudeau Sanatorium Saranac New York has reviewed (187 p 208 ff) the history of the use of

cardium in coronary artery disease and in hypertension. Congenital malformations will remain with us, and there is reason to expect that cardiovascular sound in these cases contains more diagnostic information than has hitherto been appreciated.

Thus, although the stethoscope will become less useful to the cardiologist, it can never be laid aside completely. Nor is it likely that it will soon be replaced. The human ear, particularly the educated ear, is an instrument to command the respect of all—particularly of phonocardiographers who have attempted to discover in their recordings of heart sounds information which

has escaped perception by the ear, or even to display what the ear has been able to detect. Improvements on the classical acoustical stethoscope, such as compact electronic stethoscopes using transistors, when used in combination with that excellent instrument the ear, hold much promise for the future.

Phonocardiography will continue to occupy a double role: (1) of providing permanent, objective, precisely timed records with a certain limited amount of information beyond the ken of the ear, and (2) of instructing the ear in what it should be hearing with the aid of the stethoscope.

## SECTION II

### *General and Basic Considerations*

cough to elicit rales. The earliest description he could find was by J. Gleize, who wrote in France in 1827. Barth and Roger (see p. 30) wrote an excellent text on auscultation which first appeared in 1841, went through many editions and translations, and had great influence in France in particular. In it, they emphasized the value of what we would call post-tussive rales at the apices in the diagnosis of early pulmonary tuberculosis. Furthermore, they emphasized the value of "expiratory cough," i.e., cough at the end of expiration followed by a full inspiration. Oliver Wendell Holmes in his *Boylston Prize Essay* (see p. 12) mentions the method, and Bowditch in his *Young Stethoscopist* (see p. 12) emphasized its usefulness. Edward Livingston Trudeau (1848-1915), famed American phthisiologist, probably did more than anyone else to spread use of the method in this country.

Austin Flint (see p. 29) placed great emphasis on the pitch of the breath sounds (466). The term "bronchovesicular" originated with him. He was also originator of a classification of rales which was long used. He divided dry rales into sibilant and sonorous and moist or mucous rales into coarse, fine and subcrepitant (or moderately coarse).

William Wood Gerhard (1809-1872) of Philadelphia, student of Louis in Paris, author of earlier textbooks on examination of the heart and lungs, and important figure in the differentiation of typhus and typhoid, is usually credited with pointing out that the breath sounds at the right apex are louder than at the left (342).

Only scattered notes on the history of respiratory sound are provided here, since it was found simplest to introduce a fuller survey with the discussion of each of the several phenomena of respiratory sound. See pages 473-492 for this discussion.

## CHAPTER 3

# The Nature of Sound

Sound can be defined as consisting of vibrations which are audible because of proper amplitude and frequency. More technically, sound in air or liquid at least can be defined as density waves (or alternate condensation and rarefaction) i.e. deviations slight to be sure from the 'resting' density and pressure (see Fig. 27).

If the vibrations of a tuning fork are recorded let us say by mounting a pen on one prong of the tuning fork and running a piece of paper by the pen a regular sine wave is imprinted. Since the tuning fork produces a pure musical tone of one frequency the sine wave will make a certain number of complete cycles in a second's time. For example a tuning fork which produces the note A makes 440 complete cycles in a second and the frequency is said to be 440 cps (cycles per second). Note that the amplitude of the vibration of the fork, the distance of excursion of the prongs, may vary greatly but frequency of the vibration remains the same (within limit).

The frequency at which a structure vibrates depends on the interrelationship of mass and restoring force (stiffness or spring if you will). Any structure has a natural frequency of vibration dependent on these two properties. A large mass on a weak spring will vibrate at a low frequency; in general this is a situation obtaining in the body. A small mass on a relatively stiff spring will vibrate at a higher frequency; in the body, arterial wall—distended under pressure—and bone correspond more to the latter situation. The amplitude varies mainly with the force with which a body is incited to vibrate in its natural period. However the amplitude of vibration is not entirely independent of the factors determining

<sup>1</sup> Hz is an abbreviation used in German and some other foreign writings. Used synonymously with cps it is derived from Hertz.

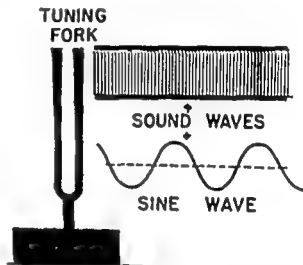


FIG. 27. Vibrating tuning fork with schematic representation of alternate areas of condensation and rarefaction and corresponding sinusoidal curve of changing pressure with reference to resting atmospheric pressure. (From Rutherford (1323).)

ing the natural frequency, the greater the mass, or the stiffer the less the amplitude of vibration and vice versa for a given force.

The characteristics of vibrations are schematically represented in Figure 28. These are the fundamental characteristics whether the vibration are those registered in the ballistocardiogram in the low frequency precordial impulse (372-400-401-818-820-1125-1409-1411), in palpable thrill (319-892) the sonic vibrations we are concerned with here or any of many other varieties.

There are three main parameters of sound (These are well demonstrated in the spectral display of sound—the sound spectrogram or for heart sound the spectral phonocardiogram). They are (1) time (2) frequency and (3) amplitude.

<sup>2</sup> Time and frequency are of course interdependent.



TABLE I

Typical intensity levels of familiar sounds

| Source   | Intensity |
|--|-----------|
| Whisper  | 10        |
| Normal conversation                                      | 40        |
| Shouting   | 120       |
| Jet engine   | 140       |
| Hydraulic press (3)                                      | 150       |
| Large pneumatic riveter (4)                              | 160       |
| Tram   | 110       |
| Chipping hammer (3)                                      | 120       |
| City street  | 70        |
| Heavy trucks   | 80        |
| Train whistle  | 110       |
| 10 hp outboard   | 100       |
| Small trucks accelerating                                | 80        |
| Light trucks in city                                     | 70        |
| Office   | 60        |
| Heavy traffic  | 80        |
| Conversational speech                                    | 60        |
| Accounting office  | 60        |
| Chicago in full trial area                               | 80        |
| Minimum levels for residential areas in Chicago at night | 40        |
| Broadcasting studio                                      | 100       |
| Broadcasting studio (max)                                | 120       |

Adapted from Beranek (58)

TABLE I-Continued

| Source   | Intensity |
|--|-----------|
| Whisper  | 10        |
| Normal conversation                                      | 40        |
| Shouting   | 120       |
| Jet engine   | 140       |
| Hydraulic press (3)                                      | 150       |
| Large pneumatic riveter (4)                              | 160       |
| Tram   | 110       |
| Chipping hammer (3)                                      | 120       |
| City street  | 70        |
| Heavy trucks   | 80        |
| Train whistle  | 110       |
| 10 hp outboard   | 100       |
| Small trucks accelerating                                | 80        |
| Light trucks in city                                     | 70        |
| Office   | 60        |
| Heavy traffic  | 80        |
| Conversational speech                                    | 60        |
| Accounting office  | 60        |
| Chicago in full trial area                               | 80        |
| Minimum levels for residential areas in Chicago at night | 40        |
| Broadcasting studio                                      | 100       |
| Broadcasting studio (max)                                | 120       |

oscillogram the waveform of musical sounds produced by musical instruments. The harmonic series are demonstrated in the spectrum and regularly spaced vibrations in the oscillogram. The presence of harmonic series is the basis for the designation of a murmur as musical.

Speech sounds show particularity in the case of the vowel harmonic pattern but not generally considered musical. What physical change occurs in the sound when a vowel is sung rather than spoken? When the sound called musical in one case non-musical in the other it is a matter of vibrato—fine rapid oscillation in frequency through a narrow range—which determines musicality in the aesthetic sense. This feature probably is missing in the so-called musical murmur nonetheless they are musical in the more general physical sense.

Except for the relatively uncommon musical murmurs most cardiovascular sound is not a composite of vibrations of pure frequency but rather is made up of vibration more or less randomly distributed over an appreciable range of frequency.

The intensity of sound in air away from sources follows the inverse square law—intensity diminishes as the square of the distance. If the distance from source to ear is doubled the intensity is likely to be reduced by 75 per cent. In bounded media (air or fluid) this law does not apply. In air-filled tube with the microphone transmits certain frequencies with little loss of intensity.

The subject of quantification in sound is general and in cardiovascular sound in particular is a complicated one. When represented in terms of sound pressure the unit of sound intensity is dyne per sq cm the meter (One bar is one atmosphere  $1.013 \times 10^6$  dynes per sq cm).



## CHARACTERISTICS OF VIBRATIONS

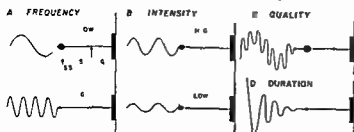


FIG. 28. *A* The frequency of vibration is determined by the relation between the mass and the elasticity of the body. As shown in the examples schematized here the larger mass vibrates at a lower frequency. *B* The amplitude of vibration (intensity) depends on the amount of displacement from the position of rest and therefore on the energy imparted to the system. *C* The quality is dependent on the modes of vibration. In the example shown two vibrating systems are connected in series. The resulting vibration is complex to the extent that there is a major mode of vibration—the fundamental—and a superimposed minor mode of vibration—an overtone or second harmonic. *D* The duration of a vibration after the source of energy is cut off is dependent on the level of the energy and the rate at which the energy is dissipated. The dissipation is greater if the frictional resistance to motion of the body is greater. (Courtesy of Rushmer (1924) and W. B. Saunders Co.)

tude (or intensity). In psychophysical terms the characteristic of duration is obviously related to the parameter of time; pitch is related to frequency; loudness, the magnitude of the auditory impression is related in a complicated way to non-linear manner to intensity (see below).

A fourth characteristic of sound is quality, known by the French as *timbre* and by the Germans as *Klangfarbe* (an expressive word meaning 'sound color'). Quality is a derived characteristic that is it is a resultant of all three of the physical parameters of sound: frequency, time and intensity. In particular, it is a resultant of the relative intensities of the component frequencies of the sound. Quality is analogous to color which is also determined by the relative proportions of light of different wave lengths (or frequencies). Just as white light can be analyzed into a rainbow spectrum of colors covering the visible range, similarly so-called white noise can be analyzed into a spectrum of frequencies covering the audible range. Just as the relative proportions of com-

ponents determines whether a color is fire engine red or deep maroon, similarly the relative proportions of frequency components determine the difference in the quality of the note middle C, let us say, as played by a violin and by a trumpet.

Musical sounds are characterized by vibrations which are perfectly regular in frequency. Usually there are several frequency components, each a regular vibration and all at frequencies which bear a simple integer relationship to each other. Each component is referred to as a harmonic. Musical sounds can be resolved into a small number of sine waves at frequencies which are simple multiples of the lowest. The pitch of a musical sound is usually determined by the frequency level of the fundamental which is the harmonic component of lowest frequency and usually of greatest intensity. The quality, however is determined by the relative intensity proportions of this and the higher harmonics (overtones or partials). (See Fig. 91B for demonstrations of harmonics in some musical sounds.) Harmonics result from the fact that vibrating structures, even in the case of musical instruments, generally vibrate not only as a whole but also in separate parts to some extent. From Figure 28C this will be evident and it will be noted that the amplitude or excursion of vibration tends to be greatest in the case of the major mode of vibration—the fundamental. A trained observer can detect by ear not only the fundamental but also one or more overtones in a musical sound (1932).

In the sound spectrogram the several harmonics of musical sounds are represented as horizontal bands at various frequency levels. In the oscillogram the regular vibrations are represented as regularly spaced oscillations in the horizontal line with the fundamental dominating as a rule. The oscillogram of a musical sound is the composite of several sine waves into which the recording can be resolved by Fourier analysis. In the oscillogram overtones with intensities less than one tenth that of the fundamental are likely to escape identification, in the sound spectrogram overtones very much weaker in relation to the fundamental are identifiable.

In the general category of cardiovascular sound there are so-called musical murmurs (see Chapter 13) which display in the sound spectrogram and

parameters since frequency is a matter of number of vibrations per unit of time (cycles per second)

mately equal to the mean pressure fluctuation accompanying Brownian motion.

Sound pressure level in db relative to 0.0002 microbar =

$$20 \log_{10} \frac{\text{absolute pressure (dynes/cm}^2\text{)}}{0.0002}$$

Particularly it should be noted that one cannot add two values in decibel directly; furthermore a noise of 80 db does not have a sound pressure level twice that with a noise at 40 db.

Practically speaking it is useful to keep in mind that as follows directly from the above formula 20 decibels represents a 10 fold difference in sound pressure; 40 db 100 fold; 60 db 1000 fold; 80 db 10,000; 100 db 100,000; 120 db 1,000,000. Doubling the sound pressure above the threshold represents a change of 6 db.

In addition, loudness is expressed in terms of the phon. However since loudness has physiological impurities as much or more than physical ones this system of unitage will be discussed in connection with the properties of the auditory mechanism.

The chart in Table I presents the intensities of certain noises familiar in everyday life.

In connection with the intensity of murmur, the *size of murmur* is a meaningful and useful concept. In evaluating the result of the Hufnagel operation (1977) for example it was obvious that the decrescendo diastolic murmur not only was reduced in peak intensity but also became shorter after operation. A measurement of the area occupied by the frequency-time (SLC) or intensity-time (PCC) plot seemed theoretically a better basis on which to compare the murmur before and after operation. It was this measurement which was referred to as the *size of the mur-*

*murmur*. (The sonoclogram of Rushmer and colleagues (1929) provides a graphic presentation of murmur size directly—see page 84.)

An important measurement is the speed at which sound travels. In air sound travels at the rate of about 1100 feet (300 meters) per second. However in water the rate is 4400 feet (about 1300 meters) per second so that in the tissues of the body a rapid rate might be expected. However this does not prove to be necessarily the case (see Chapter 11). The rate in soft tissues is probably lower and that in bone may be higher.

In music frequency or pitch is sometimes expressed in terms of octaves above or below a given pitch such as 'middle C'. In essence this is a logarithmic system. However this system is of no use in the case of cardiovascular sound (cycles per second (cps)) and an adequate—and because it is absolute—a preferred unit. The frequency range of the piano keyboard and its relation to the range of cardiovascular sound is presented in Figure 20.

Several words in the acoustic jargon should be defined briefly. *Transient* is the name indicate a brief sound. The so-called heart sounds are transient or, better in the case of the first and second sounds, at least a combination of transient. *Impact sounds* again is the term indicates are sounds of collision. Typewriter noise, footsteps and hand clapping are three examples from day-to-day experience. The valve closure sound may be in small part impact sounds produced by the actual collision of the closing cusps. *Wide band noise* means sound without harmonic pattern but with frequency representation over an appreciable pin. Most murmurs are wide band noises.

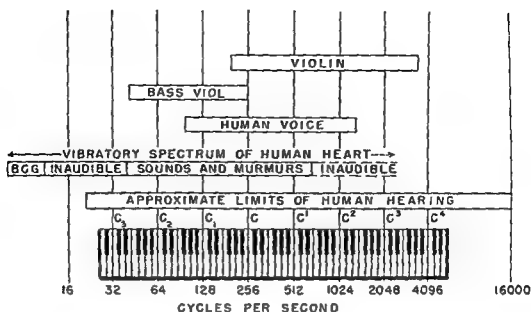


FIG. 29 Relation of frequency range of piano keyboard to that of various natural sound sources: the voice and two musical instruments. (From Butterworth (201))

dynes/cm<sup>2</sup>) Referred to is the maximum pressure (above the mean or ambient pressure) which is developed in the process of compression and rarefaction. When referred to the threshold of audibility the unit is the *decibel* (db). The sound pressure at the average threshold of human audibility (at 1000 cps) is taken to be 0.0002 microbar by international agreement. This may be considered as zero decibels of sound intensity. The decibel scale is logarithmic: a feature essential to expressing the wide range in acoustic power which the ear can encompass (in terms of acoustic power a ratio of about 10<sup>6</sup>:1 between loudest and faintest).

The decibel is ten times the logarithm to the base 10 of the ratio of two acoustic powers, e.g. if one power is 10 000 times another the difference is 40 db. (Originally the decibel scale was worked out on the assumption that the ear obeys the Weber-Fechner law strictly and that physiological loudness varies as a logarithmic function of the physical intensity. Approximately one decibel is a just noticeable difference in intensity.) The decibel is also used to express the ratio between two sound pressures. Since sound pressure is proportional to the square root of the sound power (and the amplitude proportional to the square root of the energy), the sound pressure ratio for a given number of decibels is the square root of the corresponding power ratio. In the

example given above the pressure ratio corresponding to 10 db is  $\sqrt{10\,000}$  or 100, or 20 db<sup>1</sup>.

Since the decibel system is a logarithmic one decibel ratios not absolute values, there is no zero. The value expressed in decibels is absolute only with reference to a certain level taken as the base.

$$\text{db} = 10 \log_{10} \frac{\text{sound power measured}}{\text{sound power taken as base}}$$

When the unit *decibel* is used by itself in acoustic measurements it is generally understood that it applies to the reference sound pressure generally accepted is the threshold of audibility at 1000 cps i.e. 0.0002 microbar. How small a value this is can be appreciated by realizing that this is a variation superimposed on normal resting atmospheric pressure which is usually about 10<sup>6</sup> bars. The threshold level of pressure is approxi-

<sup>1</sup> In the mind of most laymen the *decibel* means only a unit of measurement of the intensity of noise. Actually the term was carried over into acoustics from electrical communication engineering, where it was used originally and is still used principally to indicate *power* ratio.

<sup>2</sup> The reason the value is 20 rather than 10 is based on the fact that there is a squared relationship between pressure and power i.e. power is the square of pressure.

$$20 \log_{10} \frac{\text{abs. pressure}}{0.0002} = 10 \log_{10} \left( \frac{\text{abs. pressure}}{0.0002} \right)^2$$

whenever an amplifying stetho cope is used or when recordings of heart sounds are played back by means of a speaker for teaching purposes. For example, Fletcher Munson considerations enter in the process of playing a recording sufficiently loud that everyone in an auditorium can hear it; the components of lower frequency in sounds

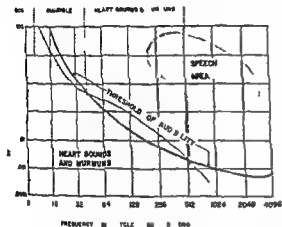


FIG. 30. Relation between frequency intensity composition of cardiovascular sound and frequency response characteristics of the human ear, specifically the threshold of audibility. (From Butterworth (204))

which are complex in respect to frequency are relatively exaggerated. As a result the sounds impress the ear as unnaturally low pitched and booming—unnatural as compared to the findings with the stetho cope. If the objective is to reproduce the stethoscopic impression arrangements for pitch control must be provided.

Several psychoacoustic peculiarities are worthy of note in connection with cardiovascular sound. A loud murmur may mask a heart sound occurring just after the murmur even though it is not superimposed on the heart sound (strictly speaking what is referred to is perhaps closer to fatigue than masking). This phenomenon can be demonstrated by playing the tape recording in reverse. A heart sound not heard on normal playing may be heard on playing in reverse or the converse may be true that is a sound just preceding the murmur may escape detection on reverse playing (204).

The difference in the threshold of hearing, depending on whether audition is binural or monural has mainly historical pertinence and interest in connection with stetho copy. As is demonstrated in Figure 32 binural audition is

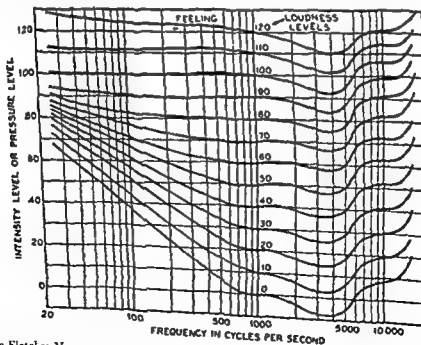


FIG. 31. The Fletcher-Munson curves (From Kerr (781)). As the overall intensity of a white noise is increased the proportionate loudness of the lower frequencies is increased. The frequency response curve for the ear becomes more nearly flat.

## CHAPTER 4

# *The Auditory Mechanism*

The ear drum is mechanically coupled to the cochlear apparatus by the ossicles of the middle ear. The cochlea is, in essence, a frequency analyzer comparable to the sound spectrograph. The human ear although in important respects not optimum for analysis of cardiovascular sound, is nonetheless a remarkable instrument. In terms of absolute sound pressure, the average normal threshold of audibility is 0.0002 dynes per sq. cm. This is equivalent to an excursion of vibration in the same range as the diameter of larger molecules and the range of Brownian motion! The sound pressure at the threshold of audibility is, furthermore, about  $1/10,000,000,000$ th of an atmosphere about equal to the weight of a mosquito wing. Some persons have sufficiently acute hearing that the noise of molecular collision in Brownian movement is audible (639). The ear drum need move only a distance equal to one tenth the diameter of the hydrogen molecule for sound to be heard; furthermore the basilar membrane need move only one tenth as far as the tympanic membrane.

All statements about the performance of the ear represent the mean of a very large number of individual testings. For any single individual no smooth curve such as those shown in Figures 30 and 31 will be obtained. Instead the curve is likely to show peaks of performance at certain frequencies and the threshold may be higher or possibly lower than that indicated and yet be entirely normal.

The average young healthy ear can detect vibration with frequencies between the lower and upper extremes of about 16 and 16,000 c.p.s. However, sensitivity varies greatly through this range. Maximal sensitivity is in the frequency range of 1000 to 2000 c.p.s. Below 1000 cycles

sensitivity falls off sharply. To be detected by ear a tone with a frequency of 100 c.p.s. needs to have a sound pressure 100 times (40 db greater than) that of a pure tone at 1000 c.p.s. Most cardiovascular sound is in the frequency range where the ear is relatively insensitive.

The changes in auditory acuity with advancing years concern principally the upper end of the acoustic spectrum. The same is true with many disease processes such as middle-ear disease and otosclerosis. Auscultatory ability is relatively well preserved and may even be enhanced through removal of disturbing or masking noises.

A further complexity of the frequency response characteristics of the ear are represented in the Fletcher-Munson curves. The lines on the chart in Figure 31 are lines of equal loudness. The lowest is the curve for the threshold of audibility and is the same curve as that in Figure 30 except that it is inverted because of different scaling. In making this family of curves, the intensity of sound at 1000 c.p.s. which was interpreted by the ear as having a given loudness was compared with the intensity which a sound at another frequency must have in order to impress the ear as equally loud. The significant feature of this chart is the fact that, at a high level of absolute intensity, sounds are more likely to be interpreted by the ear as equally loud regardless of frequency content. When the radio is played at low volume, music sounds higher pitched than when played loudly. For best reproduction music lovers must either play their recordings very loudly or preferably (?) have an amplifying and speaker system which favors the lower frequencies.

In clinical auscultation the Fletcher-Munson phenomenon probably is not a factor. However

tant. When the first of the two split components is unusually accentuated and reverberating fusion with the second component and reduction in audibility of the splitting result. (An example is the lesser audibility of normal inspiratory splitting of the second sound at the base when the aortic component is greatly accentuated in systemic arterial hypertension.) It is self-evident that splitting will be likely be detected when one of the components is of too low intensity or what amounts to the same is of lower frequency composition than is easily appreciated by ear. Because of the physiologic properties of the ear a very intense component will render the ear less

capable of detecting a second component which would ordinarily be heard and which is separated from the first by an appropriate interval. When the second component is accentuated or when it initiated a murmur splitting may be more evident.

Loudness level is psycho-acoustic matter is measured in *phons*. The phon is derived directly from the Fletcher Munson chart of equal loudness curves. The loudness value in phons of a given pure tone is the sound pressure level in db of a pure tone at 1000 cps which sound equally loud. For example a 200 cps tone at a sound pressure level of 60 db has a loudness level of 51 phon.

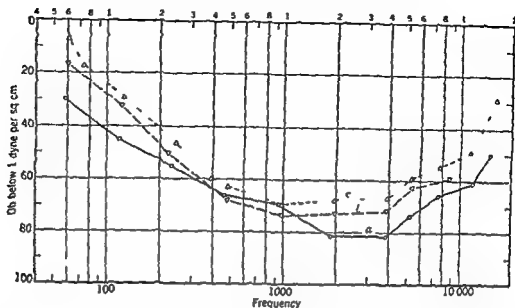


FIG. 32 — Threshold sensitivity of the human ear for binaural audition in a free field *b* binaural audition in a closed system analogous to the binaural stethoscope *c* monaural audition in a closed system (From Wever and Lawrence (1932))

appreciably more efficient (1532). The data for this chart were collected in a silent room. Under ordinary circumstances of wind and examining room the difference would be greater because of masking effects in the case of monaural audition.

Experience in phonocardiography brings to attention the fact that the auditory mechanism tends subconsciously to sort out meaningful information from that which is not pertinent from the cardiovascular viewpoint. As a result very faint sounds can be detected by ear whereas in the oscillogram, since all vibrations are recorded it may be difficult to separate out the vibrations corresponding to a faint murmur. In the spectral phonocardiogram it is easier because another parameter—frequency spectrum—is available.

Other sensory stimuli occurring during auscultation may dull auditory perception. During stethoscopy interference from other sensory stimuli should be reduced to a minimum. Astute clinicians in listening for a faint murmur not only seek as quiet a room as possible—in obvious precaution—but also take a relaxed and comfortable position and sometimes close the eyes (530). In a concert one closes his eyes to appreciate the music. The blind are often good musicians. Jeans (744) claims that pleasure information and protection are more dependent on vision than hearing. Furthermore, he states that sight developed first

in evolution and has an advantage over hearing when the two senses operate simultaneously.

The ability of the ear to appreciate "splitting" is of considerable clinical importance. How near together can two transients occur and still be interpreted as two? Using a camera shutter set at different speeds one can study this matter. The opening and closing clicks of the shutter can be distinguished as two separate sounds at as small an interval as 0.02 sec (752). Actually it is difficult to quantify minimum perceptible split since the frequency composition and intensity of the separated components are as important as the interval which separates the components. Splitting of valve closure sounds is probably less easily appreciated than are the sharp clicks of the camera shutter.

The experiments of Helmholtz and of Myer and Stumpf (744) concerning the greatest number of beats per second that can be heard has relevance in this connection. With pure tones 41 beats per second (interval of about 0.024 sec) are audible and 96 cycles 58 at 240 cycles (0.017 sec interval) and 107 at 570 cycles (0.009 sec interval). Considering that the heart sounds are complex from the standpoint of frequency, this experience with pure tones is not too disparate from that mentioned above.

Training and experience are obviously impor-

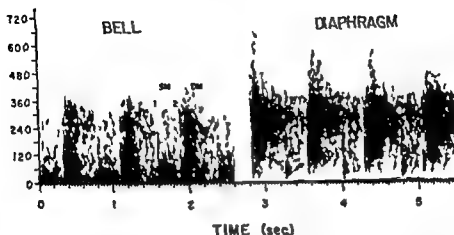


FIG 33 The time murmurs in a patient with aortic regurgitation recorded from a bell and from a diaphragm chest piece with all other conditions of recording and analyzing identical. The natural frequency of the diaphragm used in this case was apparently in the vicinity of 500 c.p.s. 1 and 2 refer to the heart sounds and SM and DM to systolic murmur and diastolic murmur respectively.

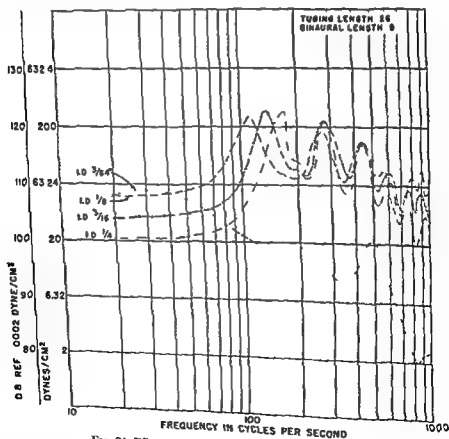


FIG 34 Effect of tubing bore on stethoscope efficiency

Response curve of a stethoscope in which the only variable is the bore of the tubing. ID = internal diameter  $\frac{1}{8}$  inch = optimum caliber of tubing (From Rappaport and Sprague (1933))



## CHAPTER 5

# *The Stethoscope*

The acoustically important portions of the stethoscope are (1) the chest piece (2) the tubing which conducts the sound to the ear and (3) the ear pieces which fit the openings of the external auditory meati.

The bell chest piece applied lightly is optimum for detection of low pitched sounds such as gullops and low pitched murmurs such as that of mitral stenosis. The only important consideration in regard to the internal shape of the bell is that the internal volume should be minimal and the cross section of the opening should be maximal. Both of these features must be compromised to some extent to allow fitting of the bell between the ribs in thin persons and in children and to avoid filling of the bell with soft tissue in obese persons. Especially at the lower end of the frequency spectrum pick up is better the larger the bell. The bell most frequently used is about one inch in diameter. This is probably about as large as is practical.

The reason for applying the bell lightly is that the segment of skin underlying the bell is in essence a diaphragm and the effect of a stiff diaphragm is obtained if pressure is applied. Many older clinicians used a bell chest piece exclusively even for the entire cardiovascular examination. (Certainly the bell is adequate and in some respects preferable for the pulmonary examination.) Probably in most instances these clinicians have found the bell adequate merely because they consciously or subconsciously applied pressure when faint high pitched murmurs were suspected.

The diaphragm chest piece selectively removes components of higher frequency. The usual thickness of the diaphragm is about 0.015 in. In

general the stiffer and the larger the diaphragm the higher is the frequency selected. There are limits to how far this can be carried, however, since attenuation of the sound, especially at the lower frequency end, always occurs and as the diaphragm becomes stiffer (see Figure 33) Some of the advantage of the diaphragm in the detection of faint aortic diastolic murmurs lies in the fact that the diaphragm chest piece is a large opening yet can have a small internal volume because the diaphragm keeps out soft tissues. Some (817) claim superiority for a diaphragm of ordinary thin photographic film. This may attain the best mentioned advantages without attenuating the sound as a stiff diaphragm is likely to do.

Yet another reason for superiority of the diaphragm in the detection of faint relatively high pitched aortic diastolic murmurs is that the intense low frequency components of the heart sounds and of any other murmur which may be present in systole for example are attenuated thereby removing a factor of masking or fatigue.

The diaphragm or the bell applied with increased pressure is useful in detecting splitting of the heart sounds. Where is at the lower end of the frequency spectrum the transients which make up the sound are intense and tend to 'run together', the separate elements are discernible at higher levels of frequency.

When in a noisy room one can easily demonstrate to himself the difference in frequency selection by the bell and diaphragm. If one shifts rapidly from one to the other by means of the valve of the 'quick change' chest piece one notes a higher pitch to room noise in the case of the diaphragm.

The tubing of the stethoscope should not be more than about 10 inches long, and the total

<sup>1</sup> See References 403, 753 and 1007

## CHAPTER 6

# The Art of Cardiac Auscultation

Within this place [the chest] we cannot see. But at this place we can listen and feel and knock, and so put it to question whether all be right beneath.

Peter Acre Latham 1847 (1843)

Auscultation requires continual practice to maintain fitness of the ear and exact perception of the heart sound just as the musician needs to practice dexterity and suppleness of the finger.

Durozier 1891 (1663)

that most important part of the total system—that between the eardrums of the stethoscope

Warren 1906 (1011)

Already commented on are the necessity of (1) avoiding interfering sensory stimuli auditory and otherwise during auscultation. (2) applying the bell with light pressure for the detection of low pitched sounds. Sometime when a very loud murmur is present removing the bell only very slightly from the chest or loosening the ear pieces so that there is a leak will attenuate the murmur and permit one to hear the heart sound proper which were previously drowned by the murmur (618 p 284). Essentially what is done by this maneuver is to introduce acoustical filtration. The lower end of the frequency scale tends to leak off to a greater extent when a light opening is present.

It is important to avoid chilling the patient especially in recording heart sounds but also to some extent in auscultation. Otherwise muscle noise of shivering may interfere seriously (Fig 491).

Ambient noise—in the ward or examining room—must be kept to a minimum for best auscultation (609). The examiner should be in a relaxed and comfortable position. He may want to close his eyes in order to concentrate better on the heart sounds (see p 66) just as one closes his eyes in a

concert in order better to appreciate the music. Obviously the patient should remove all clothing from the upper part of the body and facilities for auscultation in both the recumbent and the upright posture should be available. If one merely has the patient pull up his under-shirt one may encounter a perplexing murmur over the upper chest caused by compression of vessels at the thoracic outlet (182).

As in doing the rest of the physical examination it is a useful convention to work always from the patient's right side. A certain routine in the performance of cardiac auscultation is psychologically conducive to better performance. It is equally important to use always the same stethoscope. This was in the past more important than it is now when stethoscopes are mass produced with a high degree of identity one to another—at least among stethoscopes of the same manufacturer.

In Figure 301 is presented in example of how one should not go about cardiac auscultation. Noisy room, uncomfortable position of the physician, patient partially clothed, auscultation from the left side of the patient. Too many persons trying to listen simultaneously is another bad practice. Figure 36 illustrates the correct practice of auscultation.

Various methods must be used to keep infants and children quiet during auscultation or recording. One writer has noted that at times more information is forthcoming from slipping a lollipop into the mouth than a cardiac catheter into the vein (483). A child will often permit a careful examination when sitting on the mother's lap even though he was completely unmanageable when placed on an examining table.

Available for pediatric uses are stethoscopes

length of the stethoscope no more than about 18 inches. One must compromise convenience of use (which dictates reasonably long tubing) with the increased intensity of sounds obtainable with shorter tubing.

Tubing with an internal diameter of  $\frac{1}{16}$  inch appears to be optimal (1243), (see Figure 34). The efficiency is superior to that of  $\frac{3}{16}$  inch tubing, for example, which has in the past been commonly used. The improved efficiency is particularly evident in the range below 115 cps. Presumably the improved efficiency of the smaller tubing is the result of a smaller volume of enclosed air.

There is a high frequency cut off in stethoscope tubing (see Fig. 34) which, however, is no impediment in most auscultation. Similarly resonant peaks introduced by the influence of standing waves in the tubes probably do not introduce significant distortion.

Black tubing is professionally and esthetically most desirable.

Snug fit of the ear piece to the external auditory meatus is essential (1430). Fitting of the ear pieces should be made at the time of purchase of the stethoscope. The angle of the metal tubing of the head piece should be such that the tubes are properly directed into the ears. Since the external auditory meatus is directed anteriorly, the tubing is angulated accordingly.

Practically speaking, the type of stethoscope which incorporates both bell and diaphragm in one composite chest piece and permits quick change from one to the other by means of a valve is most useful. The chest piece is attached to the tubing in such a way that when the ear pieces are in place in the ears the diaphragm of the dependent stethoscope faces the front of the stethoscopist.

In some chest pieces the rim that holds the diaphragm in place tends to lift the diaphragm slightly off the skin. Preferable is the chest piece in which the diaphragm is shaped in such a manner that this is avoided.

Coupling is an important consideration in connection with sound, particularly in connection with stethoscopy and with the recording of the heart sounds. Even though the energy level of cardiovascular sound may be relatively high it is not heard externally with the naked ear because of losses in the transfer from the solid to the air medium. The solid stethoscope by coupling directly between ear and chest permitted conduction of sound to the ear for bone conduction. Even here there is for air conduction a coupling problem at the end of the stethoscope to which the ear is applied. In the case of the hollow stethoscope including that with flexible tubing now in use the relatively limited volume of air contained in the chest piece tubing and ear pieces is driven by the segment of skin which underlies the chest piece and functions in a manner comparable to that of the driver of a speaker.

The binaural stethoscope is advantageous compared to the monaural because (1) the ear pieces can provide a more complete seal (2) binaural audition is more efficient than monaural. In the range of 60-700 cps binaural audition is about 20 db better, a 10 fold increase in sound pressure.

No stethoscope amplifies the sound. Any advantage which use of the stethoscope has over direct auscultation resides in greater practicality plus the fact that better seal between chest and ear, binaural audition and a larger effective chest piece are possible.

manubrium and the corpus turn. The second interpiece is of course below the second rib

It will be separately indicated in the appropriate places when aicular heart disease is suspected. auscultation should include in addition to the four standard areas the following loci: (1) the base of the neck on the right (re aortic tricuspid) (2) Erb's area (third left interspace at the sternal border not so known as the second or aortic area) sitting and leaning forward (Fig. 36B) in full expiration (re aortic regurgitation) (3) the apex in the left lateral decubitus position (Fig. 36C) immediately after exercise (re mitral stenosis) Harvey and his colleagues (1932) rightly

emphasize the importance of holding down the right territorial border in cases of unexpected income reorganization.

For one reason too prolonged acclimation in one area not only is profitless but may be confusing as well. It is better to hit ten several times successively in several areas than to invest the same amount of time hitting once in each area for a longer period.

Quiet respiration can be permitted during auscultation and during recording of heart sounds as well. It can of course be necessary to have the patient suspend respiration for a time—especially if there is a question of a faint murmur. The

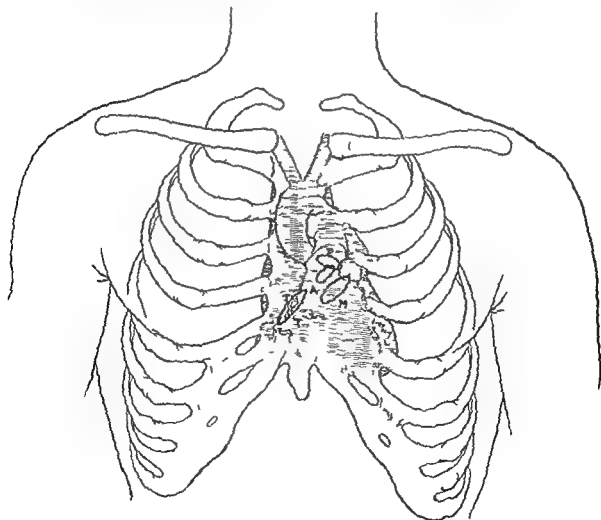


FIG. 3. The location of the heart valves with reference to the rib cage. Compare with the location as identified in the previous general classification in the valve (Figs. 60 to 64).



FIG. 35 How not to perform cardiac auscultation

Noisy, crowded room, uncomfortable position of examiner working from left side, patient partially clothed (Inspired by Harvey and Segal (1956))

precisely like those recommended for adults but with a combination bell/diaphragm chest piece of smaller size. If the bell is still too large for precise auscultation in infants one can unscrew the Bickel chest piece and listen using the metal base as a bell.

The four cardinal areas of cardiac auscultation and of phonocardiographic recording are as follows (Fig. 38).

**Aortic area**—second right intercostal space at the sternal margin.

**Pulmonic (or pulmonary) area**—second left intercostal space at the sternal margin.

**ILSB (left lower sternal border loosely tricuspid area)**—fourth left intercostal space at the sternal margin.

**Apex (apical) area**—at the apex beat or ticking an apex beat in the fifth intercostal space on the midclavicular line.

The tricuspid area is variously defined by different writers. The apex area is most variable because of the lack of clear landmarks, but the definition provided has proved satisfactory in practice.

Usually there is no difficulty in identifying the second interspace if it is recalled that the second rib is usually the highest one which is palpable with ease and that it joins the sternum at the angle of Louis, i.e. at the junction between the



FIG. 36 How to perform cardiac auscultation

**A** From right side of patient who is under stethoscope in comfortable position. **B** Listening for an aortic diastolic murmur in ILSB area. **C** Listening for a mitral diastolic rumble at the apex.

hear an extra sound usually it is best to listen for the over all center rhythm which gives the gallop its name.

In timing a murmur, whether systolic or diastolic it is useful to place the thumb lightly on the carotid pulse. There is too much delay of the radial pulse behind events in the heart to make it useful and with a very rapid heart rate even palpation of the carotid pulse may be misleading. When the apex beat is forceful it can be used for timing purposes. It is rare indeed (p. 413) that a paradoxical motion of the apex beat so-called the aortic heart beat (1887) will cause confusion.

Approximate clinical quantitation of the intensity of murmurs at the bedside was proposed by Froom and Levine (491) in 1933 using a term of grading from one to six. Grade I referred to the faintest murmur detectable. Grade VI to a murmur loud enough to be audible with the naked ear located at least a short distance from the chest or with the chest piece of the stethoscope removed at least a short distance from the chest. In more detail (888) the grading is as follows: Grade I: a murmur which is likely not to be heard on first applying the stethoscope but becomes evident on closer listening. Grade II: the faintest just audible on first applying the stethoscope. Grade III is audible with the stethoscope removed from the chest. Grade IV is the loudest murmur not audible with the stethoscope removed from the chest.

Confusion has arisen from the fact that some use a term of grading in which grades I and II refer to the first and most intense respectively. Leatham (862) suggests making a fraction of the grade in order to avoid any misunderstanding as to whether a blow of IV or V or some other number of grades is used. Thus I/VI stated grade I in VI would indicate the faintest murmur in the Levine scale.

Lepeschkin (876) has attempted to put grading on a more objective yet clinical basis by the relation of the stethoscope in which the lumen of the tube from the bell is progressively obliterated. Calibration of the size of the lumen gives the subjective impression of intensity permitted construction of a dial from which intensity could thereafter be read. Among several difficulties with this arrangement the most important was the

fact that with more intense murmurs conduction through the wall of the bell and the occurrence so that the murmur could not be obliterated even by complete closure of the tube. Another difficulty is that in adjustable tube such as that used, is a high pass acoustical filter the frequency composition of the sound as well as its overall intensity is altered. Use of an electronic stethoscope with control of the gain by means of the amplifier may represent a bedside method for quantitating intensity (879 1108 1149 1252). It is doubtful however, that the end will justify the difficulties associated with the means.

For over a century authors have used various terms for graphic representation of auscultatory findings and have recommended various terms for recording the findings of stethoscopy for future reference or the information of others. Sprague (1429) reproduced a drawing of the sounds and murmurs of mitral stenosis made in a patient's chart by Richard Cabot (p. 16). Segall (1371) has also addressed himself to this matter. In 1937 Bricker (178) published a small monograph in which a system with many excellent features is described. In recording the findings of auscultation a crude diagram can be worth a thousand words. The oscillographic phonocardiogram has never been able to fill the role. Potentially the perfect phonocardiogram might

In learning auscultation there is much to be learned for learning to associate particular sounds with particular lesions without at first analyzing minutely the components of the sound. Sir Thomas Lewis (901) wrote very emphatically on this subject using that recognition of the murmur of mitral stenosis should be a matter of learning to know it as one learns to know a dog's bark. Lewis felt strongly that instruction which urged concentration on the timing of the murmur as the first consideration resulted in incompetent stethoscopes. The instructor at first need only say: This is aortic regurgitation, why is of what makes it aortic regurgitation can come later.

Samuel Jones Gee (1834-1911) of St. Bartholomew's Hospital, early student of Celtic disease and author of a popular textbook *Percussion and Auscultation* in which first appeared in 1870 wrote as follows: Murmurs were once characterized

performance of a modified Val-salva or Mueller experiment with breath holding may occasion difficulties. Patients, particularly if dyspneic, conscious, are likely to make a great effort of "holding their breath." Since what one wishes is merely for them to suspend respiration, it is usually best to say, "stop breathing," not "hold your breath." With the latter request the subject is likely to inhale and "strum down."

Satisfactory auscultation or phonocardiography may be difficult in very dyspneic patients. The difficulties can be obviated in many such patients by having the patient hyperventilate so that he is able to suspend respiration for a time.

The influence of respiration on the heart sounds, specifically the development of splitting,

in some phases of respiration, is carefully listened for. As will be noted later, the left sternal border, particularly the pulmonary area, is the best location for detecting splitting of the second sound. Splitting occurs normally near the end of inspiration. Occurrence of splitting at another point in the respiratory cycle or persistent splitting, or splitting at the usual time in the respiratory cycle but of exaggerated degree is interpreted as an abnormality.

One should learn to concentrate on the successive time periods of the cardiac cycle separately: the first sound, systole, the second sound, diastole. With experience this becomes automatic. For detecting gallops, however, it is preferable not to concentrate too much on one element or try to

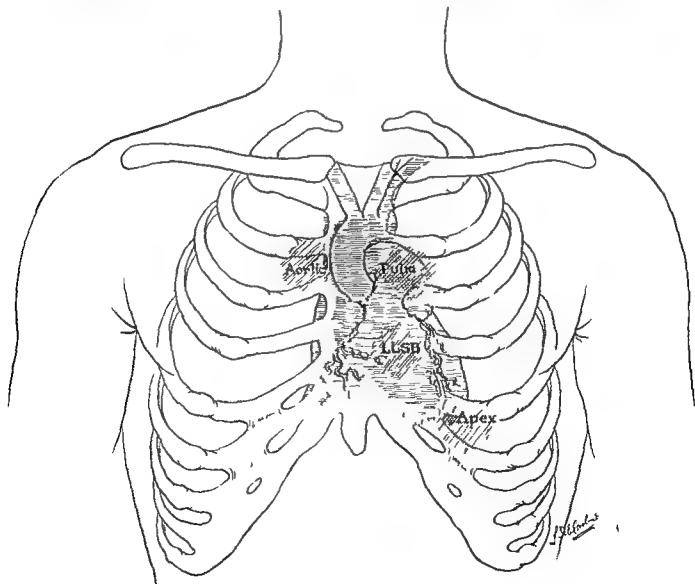


FIG. 38 The cardinal areas of auscultation (Contrast with Figure 37)

## CHAPTER 7

# Phonocardiography<sup>1</sup>

As outlined in the historical section all oscillographic phonocardiography is now done by means of electronic amplification and either galvanometer(s) or cathode ray oscilloscope with photographic camera. Spectral phonocardiography is methodologically different as far as the making of the display is concerned and particularly in the introduction of a step of analysis between the recording and the display. However common to all contemporary phonocardiography are problems associated with (1) the choice of microphone (2) the pick up of respiration pulses and other correlative and reference phenomena (3) amplification (4) artifact.

**MICROPHONES** The two types of microphones now mainly in use are (1) piezoelectric microphone (383-384) and (2) condenser (or capacitance) microphone. (The electromagnetic microphone has also gained favor with some workers and other principles such as that of variable reluctance [Waters] have been used. At present Mannheim [1033] use an electrodynamic microphone.) The advantage of the crystal microphone include inexpensiveness and relatively high sensitivity. Crystal of Rochelle salts were used exclusively in the past but barium titanate has come into use more recently. The advantages of the condenser microphone are (1) stability in respect to temperature change, moisture and jarring (2) easy calibration (3) tenability (e.g. in a hot air oven at 240° for 30 minutes). A disadvantage of the condenser microphone for recording in the operating room when explosive anesthetic agents may be in use is the presence of

a relatively high voltage between the condenser plate.

Theoretically a contact microphone has certain advantages but other clear disadvantages. The mismatch between the stiff microphone usually used and soft skin and tissue is likely to result in prohibitive loading of the skin with bothersome distortion. The microphone of Groom and his colleagues (407) is designed on the capacitance principle the skin being used as one plate of the condenser. One can even record from the surface of the heart without touching it thereby avoiding loading of the surface from which the recording is made. As a research tool it may become the standard of reference since it should provide valuable information on the precise character of the vibrations at the level of the skin. There is no air conduction of the sound, no moving parts, no loading of the skin. Room noise has less effect. The microphone is exceedingly sensitive. On the other hand hair and wax will short out the microphone and calibration is difficult or impossible.

Fortunately the use of the microphone such as the condenser type with the surface of the chest results in a much less 20 db attenuation of ambient noise. Special soundproofing is usually unnecessary for phonocardiography. However a recording area remote from elevators, the clatter in kitchen traffic in corridors and conversation of waiting rooms is essential.

Manual application of the microphone is usually superior to the use of a strap (1033). Too forceful application of the microphone is not only uncomfortable to the patient but also attenuates the sounds with alteration of frequency composition.

<sup>2</sup> This difficulty is avoided by covering the skin with light foil.

<sup>1</sup> See the Technical Appendix p 499 ff for more detail. See the Historical Section p 30 ff for background information. Also see References 501-01-06, 502-93, 1031-1106-1215.



according to their acoustic qualities, whether blowing, filing, rasping, sawing, but these are vain distinctions, in order to render murmurs serviceable in the diagnosis of disease we now regard two only of their properties: namely, their Place and Time "I cannot agree that distinctions in quality are as valuable as is commonly supposed. Many murmurs which are identical as to distribution over the surface of the chest and in the cardiac cycle are quite different in quality, and the difference in quality is a diagnostically helpful feature."

Similes are useful in teaching auscultation but care must be taken to choose familiar sounds for the analogy. The early designations for murmurs were similes: *bruit de souffle*, bellows sound; blowing murmur, *bruit de lame*, *bruit de rape*, *bruit de scie*, file-, rasp-, and saw-sounds, varieties of musical murmurs. Vivid similes have from the first enriched the literature on clinical auscultation of the heart and are an integral part of the art of stethoscopy. A keen ear for fine differences in quality and rhythm in cardiovascular sound is a valuable asset to the cardiologist. Some of the most effective teachers of clinical auscultation are those who can reproduce the sounds vocally. Similes and onomatopoeic devices are useful in teaching cardiovascular sound. The following paragraphs are quoted from a charming essay by Geoffrey Bourne (143). The piece is entertaining and instructive although many of his similes will be unfamiliar to American students.

The diastolic aortic murmur heard best in most cases down the left sternal border and to the apex has the high pitched rushing and persistent quality of water escaping under pressure from a hole over a weir or from a leaky lock gate. The diastolic murmur of mitral stenosis—not the presystolic—has the low pitched rumbling quality of a wooden wheelbarrow trundled over cobble stones. Either of these murmurs may be loud and easily heard or distant and elusive but the weir may be distant too and the wheelbarrow in the next garden but one. The quality of the sound is unchanged in both types of sound whether remote or near, bucolic or pathological.

A less rural simile is applicable to the forcible harsh, brief systolic murmurs caused by the powerful ven-

tricular expulsion of blood through a rigid and narrowed valve. Examples of this are found in aortic stenosis, pulmonary stenosis, and coarctation of the aorta. The sound here suggests the deliberate forceful and short blasts of steam given off by the Scottish express starting from rest at King's Cross. The sound may be heard from the other end of the station or from farther off still, and may vary correspondingly in volume but the character of both steam blast and murmur is very similar. When this same type of murmur is due to coarctation of the aorta it is naturally much farther thin in the other two conditions.

Another sound—a rarity—heard when an aortic valve cusp is severely ruptured, perforated, or perhaps even everted as a result of syphilis or subacute bacterial endocarditis is a diastolic croon. This murmur closely mimics the coo of a nearby and rather hoarse wood pigeon which has been restricted to one rhythmic falling note instead of the usual three or four.

The drum beat of a dance band playing a two step is a measurable fraction of time in advance of the shuffle of the dancer's feet upon a well chalked floor. The double rub of the pericardial surfaces is similarly appreciably delayed behind the beat of the first and second heart sounds. Sometimes the rhythm of the friction instead of being to and fro is triple and to modify the metaphor the dance becomes a waltz not a two step the third sound of the triplet being caused by auricular systole. Once more it must be stressed that the quality and timing of the sound are the important points. The volume varies. The shuffle of the dancer may be quite loud or if the ballroom door be shut may be heard only by close attention.

In a similar vein Bein (75) writes as follows:

*Bruit de moulin* the noise of the mill is a very happy simile since the combination of water splashing rhythmically and a background sound of wooden machinery and running water is elegantly reproduced in miniature by the sound of pneumopericardium as any knows who has listened to both.

It is in connection with musical murmurs and with murmurs audible at a distance from the chest that greatest imagination and resourcefulness has been displayed in the invention of ingenious metaphors. In part this is due to the fact that patients and other laymen in hearing the sounds have devised a simile unfettered by existing medical clichés and conventions of phraseology.

at 10 cycle has dropped a maximum of 20 db below the response level at mid above 30 cycle

One can put the reference data on the same magnetic tape track as the sound using a carrier at a frequency level of let us say 3000 cycle well removed from the range of cardiovascular sound. However, in this combination of the EKG and respiration signal with the heart sounds impairs the usefulness of the latter recording for teaching purposes. It is desirable in many situations to have a tape recorder with at least two channels so that the sound can be recorded separate from although synchronous with the other information. Of course by filtration the carrier signal at a higher frequency can be removed in a re-recording step and the sound recording recovered for teaching purposes and other audition.

In *oscillographic phonocardiography* the frequency response of the system is of great importance and the matter cannot be entirely disregarded in pictorial phonocardiography either. Rappaport and Sprague (1244) suggested two types of recordings (Fig. 40 A) one they called *stethoscopic* and represents roughly the character of the sound as they are presented to the ear by the stethoscope (there is a *stethoscopic* response curve with reference frequency response rising at the rate of about 12 db per octave) the other they called *logarithmic* because components at higher frequencies are accentuated (or the low frequency components are attenuated) in logarithmic fashion to approximate the auditory impression of the sounds (the response curve has a slope of about 18 db per octave). Recordings of the latter general type are sometime called earlike (Cer. Ohrähnliche or Chorähnliche) Leithism (839) call this type of recording, high frequency (Fig. 40 B) logarithmic. In his opinion too forbidding a term for physicians. There is in addition the so-called *linear phonocardiogram* which has minimal low frequency component. Because of the intense low frequency components in the precordial vibrations linear phonocardiograms have very limited usefulness. Many e.g., Johnston (751) and Lussada and Migri (959) have made a particular study of the

RESPONSE CURVES OF THE PHONOCARDIOGRAPH

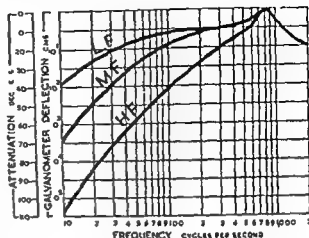


FIG. 40 A schematic representation of the frequency response characteristics of several phonocardiographic systems. (A) Leithism a high mid and low frequency system (839) B Mannheimer multiple filter system (1031) The various systems are necessitated by the fact that large intensity range of cardiovascular sound particularly the very intense low frequency components and the relatively faint higher frequency components cannot be displayed simultaneously in a single oscillogram. A second motivation is the ability to obtain approximate information on the frequency composition of heart sounds and murmurs by the examination of oscillographic displays of various frequencies band. The system of Max and Weber (1001) is similar to that of Mannheimer. In addition selective phonocardiography (993) uses a filter which cuts off the components above as well as below a certain frequency band.

low frequency precordial vibrations and Leithism and colleagues (100-101) refer to the recordings kinetocardiogram.

The logarithmic (high frequency) phonocardiogram has been useful for displaying murmurs and the stethoscopic (mid frequency) phonocardiogram for the heart sounds proper.

Unless the frequency response characteristics

\* At first the two types of recordings were achieved by the use of two microphones of different frequency characteristic later by electronic filtration.

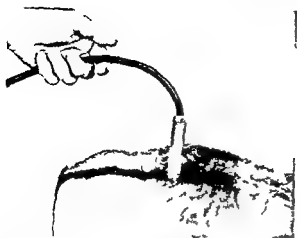


FIG. 39 This manner of holding the microphone reduces extraneous noise due to hand tremor and standardizes the pressure of application of the microphone—its weight

tion, in effect, one creates a stiff diaphragm of the skin. The pressure with which the microphone is applied is variable which should be controlled. A practical way to keep it relatively constant is by holding the microphone as shown in Figure 39. The pressure of application is the weight of the microphone and is therefore constant from patient to patient and from time to time or from area to area in the same patient. The method has the additional advantage that slight hand tremor has little effect being absorbed by the flexible cable. Unfortunately, the system breaks down when one records from the axilla since the microphone itself must be held in such a site. Clear looking, oscillographic phonocardiograms may be obtained when the microphone is held firmly with a strap or suction cup rather than lightly with the hand. This fact may be the result of attenuation of bothersome low frequency vibration by a diaphragm effect of the skin underlying the chest piece. In the SPCC because of greater dynamic range low frequency attenuation by electronic or mechanical artifices is less essential.

The presence of much hair on the chest may be troublesome. Crinkles may be introduced and it may be difficult to attain an intimate seal of the microphone with the skin. In such instances the chest can be wetted to flatten the hair and the microphone held in the usual manner. Simpler and usually more effective is to hold the microphone firmly to the chest directly with the hand. This procedure both holds down the hairs and

ensures good skin seal. Vaseline or preparations containing lanolin which are available commercially for dressing the hair have also been used with some success.

The lowest possible level of electronic noise in the amplifier is essential. Otherwise, in attempting to pick up very faint murmurs and sound, amplifier noise may predominate. A favorable "signal to noise ratio" is necessary. What the electronics experts term valve noise is particularly troublesome at the first valve. With full gain on the amplifier the "base line" of the oscillographic record should be smooth and the background of the spectral phonocardiogram clean. The microphone should, of course, be similarly silent.

Recording heart sounds on magnetic tape has great usefulness for teaching purposes and is a permanent record. Phonocardiograms, either oscillographic or spectral, can then be made as desired and other operations such as selective filtering accomplished. The ability to put reference recordings such as electrocardiogram and pneumogram on the tape also by means of frequency modulated carriers is facilitated greatly by the accumulation of libraries of recordings which can be variously studied. The techniques for analysis and display of the sounds change but the recordings are always available for re-study.

In the case of all the spectral phonocardiograms published here the sounds were first recorded on magnetic tape and the display made later. The electrocardiogram and respiratory mark will be similarly recorded on tape. Any pressure pulse, such as the jugular venous pulse or carotid pulse can similarly be recorded on the tape after transduction into an electrical signal by appropriate transducer. See Figure 40 for spectrogram made from tape recordings which used the jugular and carotid pulses for time reference.

In this laboratory most recording has been done at a tape speed of  $7\frac{1}{2}$  inches per sec. occasionally at 15 inches per sec. (Butterworth (204) has championed slower tape speeds, e.g.  $1\frac{1}{8}$  inches per sec.) Without imposed low frequency compression the response characteristics of the entire system from microphone through to magnetic tape are flat above 30 cps. At 20 cycles the response had dropped 5 to 10 db and

may be exceedingly loud. In the same patient there may be an exceedingly intense aortic murmur of aortic stenosis and a barely perceptible diastolic murmur of aortic regurgitation. For the oscillogram to record both in their true intensity proportion, the excursions representing the aortic murmur might have to be several meters high in order for the diastolic murmur to show up with an amplitude of one or two millimeters. In practice, voltage limitation is employed in such instances to place a ceiling on the aortic murmur and bring out the diastolic. The phonocardiogram by spreading out frequency and thereby reducing the intensity to be represented to that present in each frequency zone attains the problem of intensity display in a different manner and more nearly succeeds in representing sound in their true intensity proportion. Voltage limitation truncates loud murmurs and distorts their intensity time contour as represented in the oscillographic PCC. Again the PCC displays more faithfully the true shape of the murmur, a feature of no small usefulness in identifying the cause of the murmur and relating the murmur to hemodynamic.

A problem distinct from calibration although related to that of standardization in oscillographic phonocardiography. It is a hindrance objective to have uniformity of instrumentation such that recordings from different laboratories can be compared. A beginning on standardization has been made (81, 804, 1118). Until certain standards are generally accepted and given the stamp of approval by an international body such as the World Congress of Cardiology, care must be taken to define the characteristics of the instrumentation used in any serious phonocardiographic investigation.

What reference recordings are most useful for correlation with the heart sound recordings? The electrocardiogram is necessary in all cases and by its minimum standard limb lead II is usually used. The objective is to obtain sufficiently large complexes for timing purposes. Other leads must be tried when lead II shows the QRS is small in lead II. In special instances when the I wave must be recorded for correlation with atrial sound and murmurs the lead with the largest I wave must be sought.

Ossis and Braun-Mendoza (1166) and many others (1098) have championed the venous pulse (499) specifically the jugular pulse, it was their contention that the timing of diastolic events was most satisfactory by correlation with venous pulse. More recently it has been a fairly general opinion that the difficulties in recording the venous pulse and the lag between the events in the heart and the reflected changes in the neck make it objectionable for routine recording. One study (402) arrived at the conclusion probably erroneous that the physiologic third heart sound is a physiologic opening snap of the AV valve. The conclusion was based on temporal correlations of the heart sounds with the jugular venous pulse and the latter have been responsible for the results. Luo *et al.* (828) found a delay of as much as 0.14 to 0.20 sec in transmission of the venous pulse wave to the jugular vein.

Another objection to use of the jugular pulse is the technical difficulty of recording especially if cups or timbours are used. Piezo-electric transducers can be applied directly to the overlying skin. Furthermore, Haldack and Wolf (706) have made use of a recording device consisting of a light paper arm or 'rider' which is applied to the skin overlying the jugular vein and which interrupts to a variable degree a beam of light passing from a source to a photoelectric cell.

Leitham (849) has emphasized the usefulness of the indirect carotid pulse tracing. Particularly is the arterial trace helpful in the epiratic identification of the aortic and pulmonary closure sounds—a matter of considerable clinical importance by the evidence of Leitham. One must allow for a delay of 0.02–0.04 sec part of which occurs in transmission of the pulse to the neck part in the recording device. If a cup is used for the pick up of the carotid pulse application of the cup is critical (706). If improperly applied one may actually obtain a wholly or partly inverted pulse curve. An atrial dip may leave one uncertain as to the point at which one of the arteriatic limb should be placed. The contour of the pulse is recorded from a somewhat haphazard application of a cup or timbour type pick up is not reproducible and cannot be depended upon to give

of the oscillographic recording system is satisfactory, errors in display may be produced, for example, the presystolic murmur of mitral stenosis may not seem crescendo because the higher frequency components which make a contribution to the total intensity and increase with the crescendo are not properly represented. For a similar reason the systolic murmur of aortic stenosis may not be properly diamond shaped. At this point a plea is made for all workers publishing results of phonocardiography to determine and report the frequency response characteristics of the entire system used.

The frequency parameter has been manipulated in other ways. Minnhemer and Stordal (1931) by selective filtration split the sounds into five separate although overlapping frequency bands (Fig. 40), representing the information in each band as in oscillogram. His bands were as follows: 0-100 cps, 50-175, 100-200, 175-400, 200-500, 500-1000. Contrary to what is generally thought, the filters used by Minnhemer attenuated the signal to a variable degree only at the lower end of the frequency spectrum (see Fig. 10 C). The output from the five filters was recorded in a parallel manner by a multi-channel recorder. This method was termed 'calibrated phonocardiography', features of intensity calibration are discussed later. Maass and Weber (1901A) specified a multi filter multi channel phonocardiographic system comparable to Minnhemer's and Holldack and Wolf (706) make extensive use of a system which similarly splits the sounds into five frequency bands in roughly the same range. Linsadi (993) has used what is, for practical purposes, the same method but which he calls "selective phonocardiography". Linsadi does make use of both high and low cut filtration, that is, he attenuates the components of the signal in a frequency range above as well as below a specified range.

Junggren (959) provided a critical appraisal of the Minnhemer-Stordal system which in essence consisted of crystal microphone pre-amplifier filter system amplifier recorder. He pointed out the risk that distortion in the form of *de novo* generated overtones might be introduced at the pre-amplifier stage because of overloading by high amplitude vibrations in the lowest

frequency band (0-100 cps). This distortion would have the result that in the recording, from higher frequency bands components which do not actually exist would appear too long a time for build up and for decay of filters may introduce time distortion. The important considerations must be kept in mind in any system involving filters including spectral phonocardiography.

The oscillographic phonocardiogram can be registered by means of galvanometers or the cathode ray oscilloscope. In the case of both a phototriple record can be produced. Direct written records are possible in systems using galvanometers. Holldack and Wolf (706) and others (1325) have used direct writing galvanometer recorders with seemingly satisfactory results. Direct writers with excellent frequency response characteristics (e.g., the jet ink writer of the Helmut Co.) are now available.

Intensity calibration in phonocardiography presents many problems (886) there are variables in the sound transmitting properties of the chest among patients and in the same patient under different circumstances (see p. 147 ff). There may be a variability in the performance of the microphone which often is not easy to calibrate in the first place. The electronic amplification must be quantitated. Complicating matters greatly is the fact that changes in intensity must be considered in relation to frequency. Sounds of quite different frequency composition may be of identical overall intensity.

For calibrating purposes Linsadi and Gamma (983) use a sound generator strapped to the chest in about the left anterior axillary line. McGregor, Rippaport Sprague and Friedlich (1066) attempt to calibrate only what reaches the surface of the chest and in fact only what came out of the microphone. An electrical calibrating signal was introduced proximal to the microphone. Storm and Greer (1398) used a similar method by introducing at the input stage a standard signal at 30 cps and 20 db above the threshold of audibility.

The dynamic range of cardiovascular sound is enormous. In the extreme instance the loudest sound is painful to the ear and the faintest is located at the threshold of audibility or below. In congenital heart disease in children the murmurs

mended from time to time and are used especially for investigative purposes. Obviously recording indicated from any area where stethoscopes indicate a sound of interest to be located.

Duchosal suggested a system of labeling recording using letters and numbers e.g. 2L3 = second left intercostal space 3 cm from the mid-axillary line. M = mid-sternal at level of fourth intercostal space. Some may find it useful to have printed form or a rubber stamp, so that by means of a number the precise site of each recording can be indicated during the recording procedure.

Recording from the esophagus can be made either by attaching the transducer to the end of a catheter or by having the subject swallow a small microphone (68). By either method it is desirable to insure contact with the wall of the esophagus by means of inflation of a balloon with water or air. An atrial heart sound is always discernible by esophageal recording unless of course atrial fibrillation or other dysrhythmia such as a premature atrial systole is present. The heart sound is recorded from the esophagus at a lower pitch than those recorded at the surface of the chest. This fact has raised the question of whether the thoracic cage may not be excited to vibrate in its own natural period and thereby contribute higher frequency components to the heart sound. Such (13374) attached his microphone to the external end of an esophageal tube 70 cm long and with an internal diameter of 3 mm. A lateral opening 10 cm long, covered with a thin rubber membrane was provided near the internal end. The diastolic murmur and especially the presystolic (Systolic) murmur of aortic valve disease was louder in the esophagus although the systolic murmur was louder at the precordium. In one case of presumed mitral regurgitation the murmur was demonstrable only in the esophagus.

Recording directly from the surface of the heart and great vessels has been difficult

\* This method of esophageal pick-up dates at least from 1891 when Hoffman and his associates (Richardson (1872) in England) inserted a catheter into the esophagus and listened at the external end. In 1915 Levin (911) used the same method of esophageal auscultation. It is of note that with this he heard an atrial sound in only one of 25 subjects.

because of the almost perpetually moving surface with possible introduction of vibrations which are not of physiologic significance and because of loading of the surface by the microphone. Bertrand Milne and Hornick (96) used suction to hold the microphone on the heart. Lepley et al. in 1939 allowing the microphone to rest on the heart and ride with it may be satisfactory depending on the area from which the recording is being made and the type of microphone available.

Intracardiac phonocardiography can be said to have had its crude beginnings with the intracardiac pressure recordings of MacLeod and Cohn (101a) who found high frequency vibrations equivalent to the heart sounds superimposed on pressure recordings. Dock (35) took delight in pointing out that the vibration corresponding to the first sound had maximal amplitude in the vicinity of the AV valve. Recording of the rapid pressure transients within the heart has been attempted by means of a barium titanate (89) or miniature electromagnet (1121) transducer on the tip of a catheter, a catheter microphone with one pole at the tip of the catheter and the other the body itself (196 (397)) or a transducer located on the external end of the catheter and communicating with the interior through the blood filling the catheter (979). If one uses an internal transducer, a double lumen catheter permits combination of intracardiac phonocardiography with cardiac catheterization for purposes of blood sampling and pressure recording. The information obtained by means of a transducer at the external end of a catheter must be interpreted with utmost caution because of distortion unknown and known. This laboratory has been disappointed in the performance of the available intracardiac transducers of the barium titanate type. Disturbances due to bending of the catheter to striking it at a distance from the tip and even to minor temperature changes and relatively insensitive to localized pressure changes at the tip they are not perfect true indicators of such frequency pressure fluctuations within the heart. When all factors are considered the use of an external transducer on a venous or arterial

(11) - Lepley et al. used the Statham P23D strain gauge.

dependable diagnostic information, as in cases of aortic stenosis. It is true that timing not contour, is the information sought in the use of carotid pulses in phonocardiography. A modification (203) of the carotid pulse tracer of Duchosal (379) avoids some of the technical difficulties of the cup type of carotid pick up and provides more meaningful records from the standpoint of contour. Around the neck is placed a collar consisting half of elastic "mole-skin" and half of a narrow inflatable cuff which is maintained at a pressure of about 20 mm Hg during recording. The interior of the cuff communicates with a Statham strain gauge. With this system it is difficult or impossible to record the venous pulse simultaneously.

Respiratory phase should also be indicated in recordings. Effects of respiration on the splitting of heart sounds and on the intensity of certain murmurs is of great diagnostic significance. For recording respiration, belts around the chest with mechanical or strain gauge pick up and face masks with various pick up devices such as a hot wire variable resistance have been used. Holl dick and Wolf (706) use the same method as for venous pulses (see above). A simple and reliable method is to mark respiration manually, i.e. watching the breathing, to turn a line up and down on the record in phase with respiration. Ordinarily there are three phases of respiration: inspiration, expiration and apnea (in this sequence). At more rapid rates of breathing the first two phases may not be interrupted by an apneic period.

In the respiration trace in the recordings presented in this monograph upward movement of the line indicates inspiration, a downward movement expiration and a horizontal position of the line apnea. (The reader should not be confused by the fact that often inspiration has been marked 'insp' at the peak (i.e. end) of inspiration and expiration marked 'exp' at the end of expiration or early in the apneic period.) In many of the recordings, electrocardiogram and respiratory trace have been combined, i.e. the base line of the EKG has been moved up and down to indicate respiratory phase.

For purposes of special investigations other reference recordings are used such as intracardiac pressures obtained by cardiac catheteriza-

tion (570, 571), apex beat (175, 176, 828), electrokymogram (12, 17), and so on. The apex beat has been useful in defining the electrical mechanical interval in mitral stenosis (see Fig. 284) and the validity of the interval so indicated is confirmed by correlation with ventricular pressures (778). Correlation of murmur shape and intensity with the pressure gradient across valves is now possible in laboratories where right and left heart catheterizations are being performed. Correlation of murmurs with flow, especially velocity of flow, would be of interest and probably will be possible in the not distant future.

The aspect ratio—the ratio of the horizontal and vertical axes of the record—is an important feature in phonocardiography. Einthoven introduced the paper speed of 25 mm per sec for electrocardiography; this time base has continued to be standard for the EKG. Phonocardiography requires a faster paper speed, i.e., a time scale which is "spread out" more for optimum definition. Flexibility in manipulation of the time scale is desirable at least in a research instrument, although it may not be essential to a clinical tool. In special phonocardiography, as practiced by means of the original model one second was represented by about 44 mm on the horizontal axis. This display although determined fortuitously by the commercial design of the sound spectrograph modified for phonocardiography proved satisfactory for routine work. However, a flexible time base is being incorporated as a feature of recent models.

**RECORDING LOCUS.** The standard areas for recording are: *a* for auscultation *aortic* (second right intercostal space), *p* *pulmonary* (second left intercostal space), *L* *LSB* (left lower sternal border, in the fourth interspace) and *a* *apex* (region of apex beat or, if none is present the left medioclavicular line in the fifth interspace). Simultaneous recording from multiple loci has been developed to a high degree by Leatham (859). The principal usefulness has been in demonstrating the normal constitution of the heart sounds in the several cardinal areas of auscultation and in aiding the identification of the origin of certain components of the heart sounds in pathologic conditions.

Several areas for recording other than the cardinal areas of auscultation have been recom-



FIG 41A A highly schematic representation of the principle of a sound spectrograph which in this instance would provide direct (i.e. instantaneous) analysis and display. (In the example of heart sound used here the normal young subject demonstrates a third heart sound present only during inspiration. There is also sinus arrhythmia.)

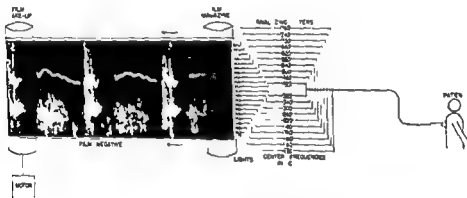


FIG 41B Same as Figure 41A except that lung sound is displayed. Harmonic represented here as horizontal band in a third. Note the much prolonged expiratory phase with a prominent harmonic.

But since fetal phonocardiography is the study of heart sounds in the fetus, spectral phonocardiography might indicate comparable studies in ghnot. The alternative designation spectro-phonocardiography, although possibly more accurate etymologically, is too cumbersome for ordinary use.

In effect the principle of the spectral analyzer is this (see Fig 41). One can think of this method as putting the sound (in electrical form of course) through a large number of electrical filters, each of which is tuned to a different and specific frequency pass band. The output of each filter could activate a tiny light. The play of the bank of lights on the moving film would in effect be the spectral phonocardiogram. Note that time is on the horizontal axis as in all physiologic recording. The vertical axis is frequency scale not in-

tensity as in the oscillographic recording. Intensity in the SICC is represented by density of a given area of the recording, for example in the schematic example used in this explanation note that the intensity of each of the tiny lights—and the density of the mark it makes on the recording medium—is a function of the energy in each frequency band of the sound being analyzed.

In practice (see Fig 42) because of the expense and other difficulties of a large number of individual filters the method used is in general terms of the type. The segment of sound which by aural editing is selected for analysis and display is played over from the original tape to the magnetic margin of a disk mounted on the same axis as the kymograph drum on which the records are made. The analyzer itself is in essence a single pass-band filter the tuning of which is changed

ten times in the oscillographic recording. Intensity in the SICC is represented by density of a given area of the recording, for example in the schematic example used in this explanation note that the intensity of each of the tiny lights—and the density of the mark it makes on the recording medium—is a function of the energy in each frequency band of the sound being analyzed.



catheter or on a rigid needle introduced directly into the chambers of the heart may, with care, provide more accurate information.<sup>7</sup> At present there is no accurate information on the magnitude of the sound pressures in the heart. The information in the next paragraph must be interpreted cautiously in light of these numerous difficulties.

Intracardiac phonocardiography, like esophageal phonocardiography, demonstrates an atrial heart sound in all cases unless atrial systole is absent. It is recorded loudest in the atrium and less clearly in the ventricle. There is a striking restriction of sound to the vessel or chamber in which it is generated. Recordings of the left side of the heart have been made in connection with "left heart catheterization." There is as much as a 100 fold attenuation of sound generated in one ventricle and detected in the other and an attenuation in excess of 60 db between the interior of the heart and the surface of the chest. The first sound is much less intense in the vicinity of the tricuspid valve than in the vicinity of the interventricular septum (89) suggesting that the tricuspid valve may be a poor noise maker. What can be interpreted as a physiologic opening snap of the AV valves can be recorded from the interior of the heart (979). In the first portion of the pulmonary artery (but not the aorta) a systolic murmur is recorded in all cases. It is possible that a systolic murmur is indeed, always present in this vessel at the peak of systolic ejection. In direct phonocardiography a systolic murmur is always found over the base of the pulmonary artery and never over the aorta (1307) a corroborative finding. However there is the additional possibility—in fact virtual certainty—that the presence of the catheter favors the development of a murmur because on fluoroscopy violent whipping about of the catheter is seen. Often when the catheter microphone has been passed into the pulmonary artery or aorta from the ventricle a faint decrescendo diastolic murmur is recorded. Presumably this is caused by regurgitation at the arterial valve which is held open by the catheter. The murmur of mitral regurgitation is loudest in the left atrium. The murmur of mitral stenosis is loudest in the left ventricle.

<sup>7</sup> Careful investigation of the frequency character-  
istics of catheter recording systems such as those by the  
Copenhagen group (636-637-1507) are pertinent.

Potentially intracardiac phonocardiography will tell us much about the sounds we hear or record at the surface of the chest. However, it is unlikely to become a routine procedure, even in cardiac catheterization laboratories, simply because it is unlikely that the information afforded will be sufficiently superior to that obtainable at the surface of the chest to make the increased risks and technical difficulties worthwhile.

Rushmer and colleagues (1325, 1328-1329) have developed a technique for producing what they call *soniclograms*. In essence these are the intensity envelope of the sound. This is accomplished, first, by half wave rectification of the oscillographic phonocardiogram, and second, by electrical integration. The resulting curves can be used for timing purposes and possibly the shape of murmurs will be useful in making hemodynamic correlations.

*Spectral phonocardiography* (Fig. 41 to 43) is the application to phonocardiography of the method of sound spectrography developed at the Bell Telephone Laboratories in the early 1940's. As in the case of all phonocardiography a display of the heart sounds is provided. The record can be either direct written (on electro-sensitive paper) or photographic. The initial recording of the heart sounds together with reference recording is made precisely as for conventional phonocardiography, that is the recording can be made directly to the magnetic recorder of the display unit or and this is for several reasons the preferred practice the information can be first recorded on magnetic tape. There is then an additional step in spectral phonocardiography between the recording of the sounds and inscription of the display—the frequency analysis.<sup>8</sup> The finished spectrogram is a time frequency intensity plot. The analysis to which the sounds are subjected is in many ways comparable to that which the ear makes. This method is called *spectral phonocardiography* because although it is a type of phonocardiography it differs from the conventional oscillographic variety in that the frequency spectrum of the sounds is portrayed. 'Spectral phonocardiography' may not completely satisfy the philolo-

<sup>8</sup> The publications of Burger and colleagues (190-196) illustrate the more laborious and non-graphic methods of frequency analysis—that of Fourier and that of Labrousse.

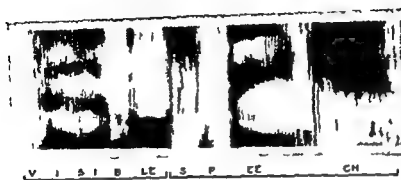
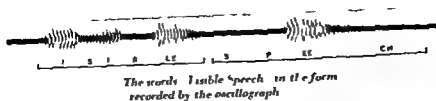


FIG. 43 From Potter et al (1230)

ferred because of (1) flexibility in manipulation of the time scale and (2) good density detail for representation of intensity.

Another filter system in principle distinct from the heterodyne method has been devised by Huggins—the phase filter (722, 1514). Although this results in improved definition and more accurate representation of the harmonic of mitral murmur (Fig. 43) noise sound which represent the majority of those generated in the cardiovascular system are demonstrable as well in our opinion. Transients (e.g. the heart sound) are poorly defined in the phase filter record.

In the preface (p. vii) certain of the virtues of spectral phonocardiography were enumerated. Figure 44 contrasts the oscillogram and the spectrogram of speech sound. There is sufficient additional detail in the spectrogram to permit one, with training, to read what is being said. This is of course impossible from the oscillogram.

In its application to cardiovascular sound sound spectrography required several methodologic adaptation. (1) Imprecise determination of the most satisfactory width of the frequency band for individual scanning and (2) devising

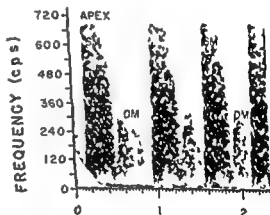


FIG. 43-1 Recording of a noisy murmur made with the phase filter (1101)

of method for incorporating the electrocardiogram and other physiologic events in the finished record. Other problems included determination of the best frequency span for routine use and the best aspect ratio (i.e. ratio of vertical to horizontal axis) (see p. 95). Also the question of whether a logarithmic frequency scale might be advantageous required an answer.

With the wide pass bands there is abrupt and early onset and a minimum of ringing so that

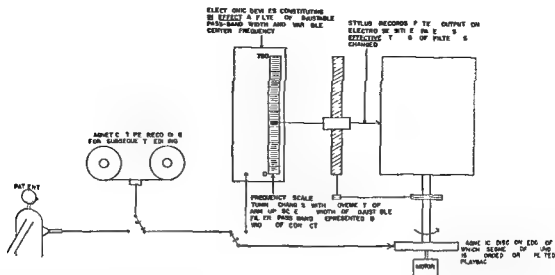


FIG. 421. Schematic representation of the sound spectrograph used in making most of the spectral phonocardiograms presented in this monograph. The intermediate step of tape recording and the serial rather than simultaneous frequency analysis are shown.

progressively as the segment of sound is played back repeatedly through it. For example during the first rotation of the magnetic disk and the corollary drum, the information in the 15 to 20 cps band might be inscribed on the record; with the second rotation the information in the 20 to 25 cps band, and so on from bottom to top of the record until the entirety of the frequency scale desired has been scanned. Actually there is much overlap of the individual frequency bands. In making an analysis from 0 to 750 cps the sound may be passed through the equivalent of 19 many as 425 individual filters each with its pass band overlapping others but with its center frequency about 18 cps removed from the center frequency of the filter above and below (see Fig. 43). It must be granted that the use of a single variable filter has the theoretical disadvantage that the Q or characteristic with respect to ringing varies with frequency. The temporal error increases at higher frequencies. This factor is, however, of doubtful significance in spectral phonocardiography.

The filter set up is of the heterodyne type and can be compared to the tuning arrangement of the ordinary radio, which also uses the heterodyne principle in turning the dial for tuning in stations one changes the tuning of the oscillator in the radio set. When the oscillator is in perfect resonance with a particular frequency the sounds at that frequency are picked up. Only a station

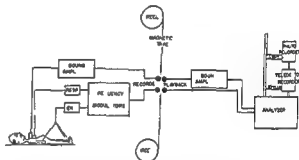


FIG. 42*B*. Schematic representation of spectral phonocardiography at stage of double display unit. The heart sounds are first recorded on magnetic tape. LKG respiratory phase and other physiologic events are recorded simultaneously on tape by means of frequency modulated carriers. The tapes are subsequently audited and sections selected for analysis. At the stage of evolution of the analyzer and display unit represented here simultaneous direct written (*below*) and photographic (*above*) records were made.

operating in the same frequency band as that at which the set is tuned will come through clearly

Note that the information can be put on a record in several ways (Fig. 43). The output of the filter can be used to activate an electric spark which marks electro-sensitive paper. Electro-sensitive paper has the disadvantage of a rather poor intensity depth. Or the output can modulate a tiny light which is made to play on photographic paper. Finally the output can be put into a cathode ray oscilloscope with a long persistence face and the image photographed. The last method is pre-

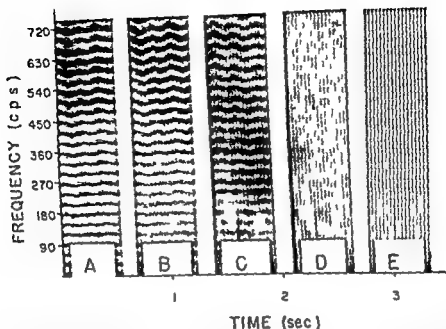


FIG. 46 Analysis through the five filter system characterized in Figure 45 of a signal with a repetition rate of 30% sec and harmonic at each 30 cps. Note that pass band C provides the best compromise resolution of both time and frequency. (Note that the artifact—noise or noise—due to irregular rotation of the drum becomes more striking at higher level of frequency because the excursion of noise is a percentage of the frequency at a given level.)

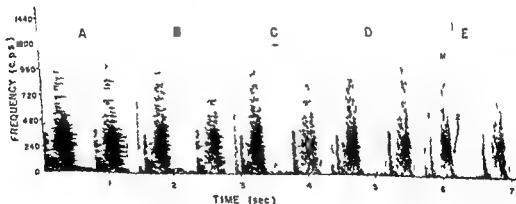


FIG. 47 The crescendo late systolic murmur displayed by the five filter system. Pass band C is considered the best compromise for routine use in the study of cardiovascular sound.

transient are sharply displayed but of course the frequency detail is by definition inferior. With narrow pass bands the logarithmic activation tend to be greater and ringing occur. The record made with wide pass bands look tracked in the vertical direction those made with narrow filter look tracked in the horizontal direction because of the manner (see Figure 45 to 48).

The instrument originally available to us commercially had a relatively wide and a relatively narrow filter system neither of which was satisfactory. A filter of intermediate width (21 cycles wide at an attenuation of 6 db from peak performance) proved to give most satisfactory compromise definition of both time and frequency. There is a difference in onset time of as much as

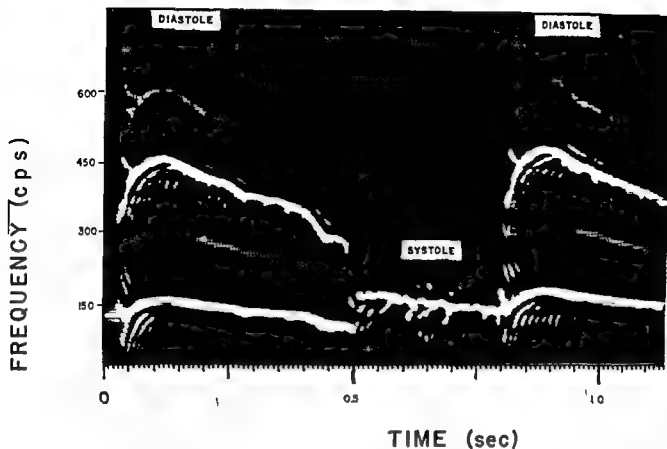


FIG 44 B Recording of a musical murmur made with the phase filter. The phase filter is unsatisfactory for the analysis of noisy murmurs. It is superior to the heterodyne system for sharp definition of the harmonics of musical murmurs. This superiority is of questionable practical value, however.

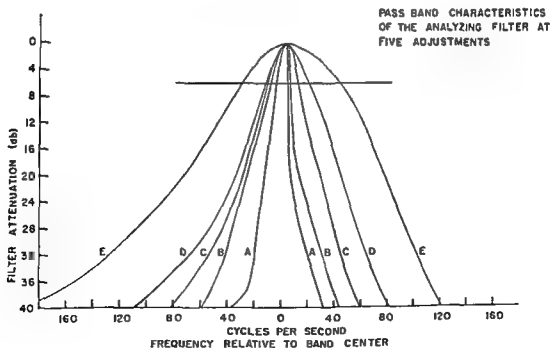


FIG 45 The pass band characteristics of five filter setups tested to determine empirically the optimum system for study of heart sounds. Defined in terms of width at 8 db attenuation (indicated by the horizontal line) the curves are A = 9 cps, B = 16 cps, C = 23 cps, D = 31 cps, and E = 71 cps.

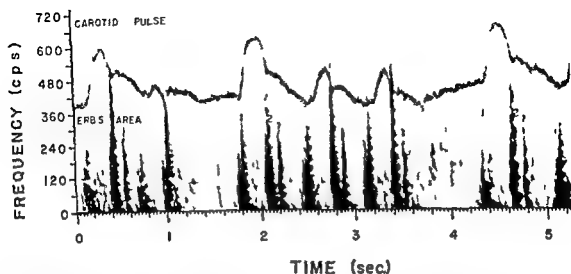


FIG. 50A Correlative recordings parallel with the spectrogram

Although the I KC was used in most of the studies (it was put on the tape as an FM signal and di played directly on the final recording as part of the frequency analysis) any pressure pulse in electrical form can be similarly recorded and di played e.g. the carotid pulse (A) and the jugular venous pulse (B). C.B. (1932) 37 saw of 1 min ha. chronic heart failure with atrial fibrillation. Three years previous to this recording a cerebral embolus occurred. The extra sound is probably third heart sound although it shows more variation with the duration of the previous diastolic interval than is usual. The alternative possibility is that it represents a mitral opening snap.

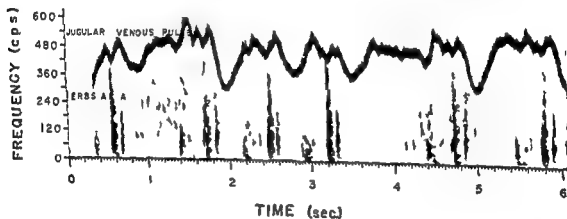


FIG. 50B See legend for Fig. 50A

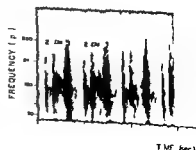
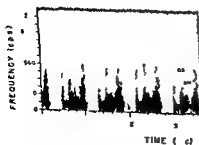


FIG. 51 Sound of mitral stenosis di played with linear (left) and logarithmic (right) scales

0.015 sec between the widest and the narrowest filter systems (A and E in Fig. 49). The lag in the filter system routinely used probably does not exceed 0.01 sec.

The EKG and other low frequency physiologic data (Fig. 50) are recorded on magnetic tape simultaneously with the initial sound recording. This is accomplished by means of frequency modulated

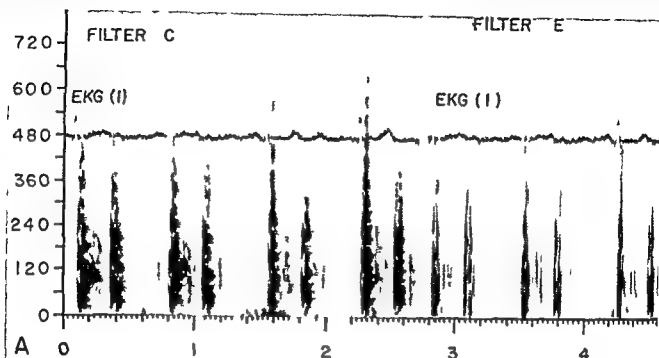


FIG. 48. Pulmonary area in normal 14 year old patient. The separate components of heart sound, specifically slight splitting, are best demonstrated by the wide filter system (E).

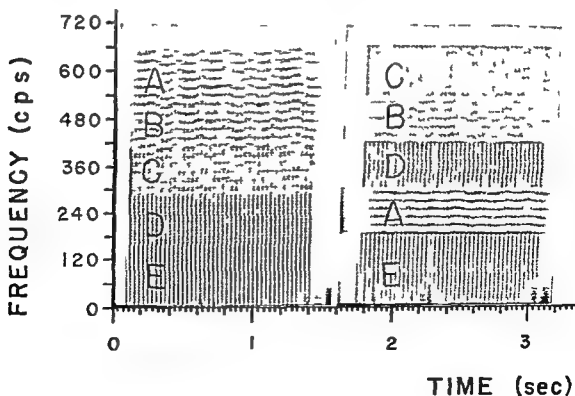


FIG. 49. The relation of filter characteristics to time lag.

The widest filter (E) has the least lag and the narrowest filter (A) has the most lag.

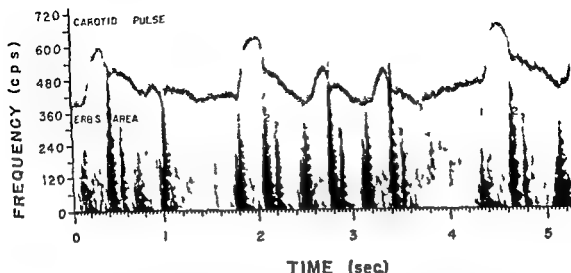


FIG. 50A Correlative recordings parallel with the plectrogram

Although the FKG was used in most of the studies (it was put on the tape as an FM signal and displayed directly on the final recording as part of the frequency analysis) any pressure pulse in electrical form can be similarly recorded and displayed e.g. the carotid pulse (A) and the jugular venous pulse (B). C.B. (410309) - severe old man, chronic heart failure with atrial fibrillation. Three years previous to this recording a cerebral embolism occurred. The extra sound is probably third heart sound although it shows more variation with the duration of the previous diastolic interval than is usual. The alternative possibility is that it represents a mitral opening snap.

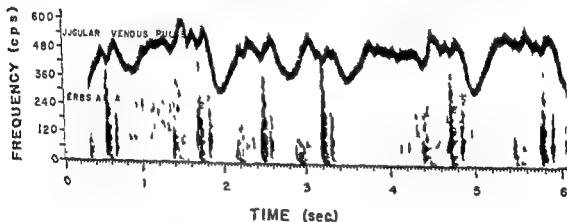


FIG. 50B See legend for Fig. 50A

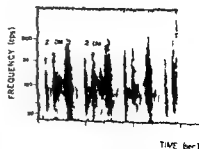
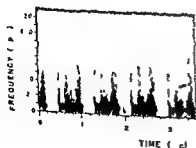


FIG. 51 Sound of mitral closure displayed with linear (left) and logarithmic (right) scales



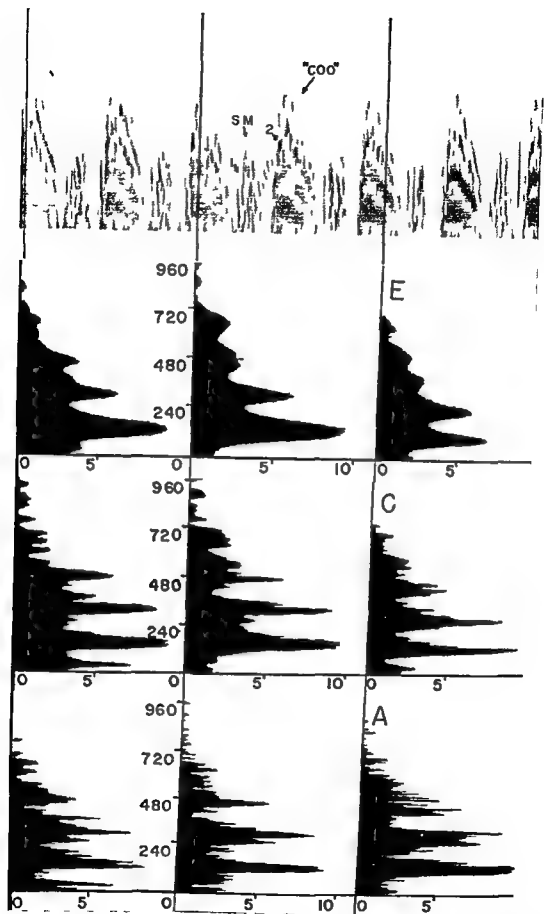


FIG 52 Intensity scanning

At the top is the spectral phonocardiogram of a cooling aortic diastolic murmur. A systolic murmur is also present (filter I). Below are preented frequency X intensity section taken at three specific points in the diastolic murmur (right to left) and analyzed through three filter systems which from above down are filters I, C, and A.

carriers. For example the FKG may be put on at 5000 cps. The amplitude of the EKG is used to modulate the frequency level. When this information which is in terms of frequency is put into the frequency analyzer an EKG or other curve of conventional appearance is yielded. By the use of frequency modulation one has in essence changed the electrocardiogram into a sound. The same procedure is used in transmitted electrocardiograms over telephone lines, but in that application demodulation has to be used to get the record back into conventional form (1240).

The rotation of the disk with the magnetic recording on its margin and of the kymographic drum which rotates on the same axis with the disk must be uniform. Otherwise wobble or wow results. For example if there is a per cent variation in the rate at which the drum rotates there will be a per cent variation in the frequency level of a constant pure tone. In the case of low frequency sounds and particularly of noises the problem is of less importance than it is in connection with high frequencies, such as the electrocardiogram. In the FKG 'wobble' shows up as a bump which should not be there. Only if they are very marked will they interfere with the QRS, however they may render the P and T wave useless for correlative purpose.

Preliminary survey made it clear that a frequency pin to 750 cps would be adequate for the great majority of applications in cardiovascular sound. This finding is consistent with that of Williams and Dodge (1956).

The aspect ratio available in the commercial model of sound spectrograph was satisfactory in our opinion. This provided height less than 2 inches per sec on the time scale and 200 cycles per inch on the frequency scale. The paper speed was approximately 44 mm per sec and the standard FKG paper speed is 25 mm per sec.

There are occasions when stretching of the time scale is desirable. With the drum method this could be done only at the expense of the frequency display, that is, the change in the speed of the drum necessary to stretch out the time scale by a factor of 2 reduced the apparent frequencies (the height of the frequency scale) by a factor of 2. In brief the drum permits little latitude in the manipulation of the time base. The cathode ray oscilloscope, a great improvement in this respect



FIG. 31 A Three dimensional model

By cutting out and stacking a large number of ten cps X frequency sections a three dimensional model can be constructed such as this one of the same as aortic diastolic murmur as shown in Figure 32. Note the curvature in the harmonic. In general the results do not justify the effort.

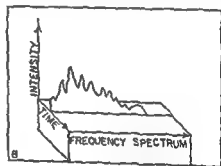


FIG. 31 B Schematic drawing of Figure 31 A

Because most of the information in cardiovascular sound is under 200 cps it seemed possible that a logarithmic scale (Fig. 31) or at least a scale with the lower frequency range expanded would be valuable. Our only exploration of this possibility seemed to indicate that a logarithmic scale has no particular advantage. However the analysis below 60-100 cps leaves much to be desired since it is lacking in detail and the logarithmic scale by giving emphasis to this zone of frequency actually seems less informative than the linear record. It may be premature to discard the expanded low frequency scale. Improvement in the low frequency analysis is being worked out and a logarithmic scale may be considered optimum in the future.

#### ARTIFACTS IN PHONOCARDIOGRAMS

PHYSIOLOGICALLY NON-CARDIOVASCULAR NOISES (Fig. 34 and 35) Breath sound, hiccoughs and muscle noises are undoubtedly the principal inter-

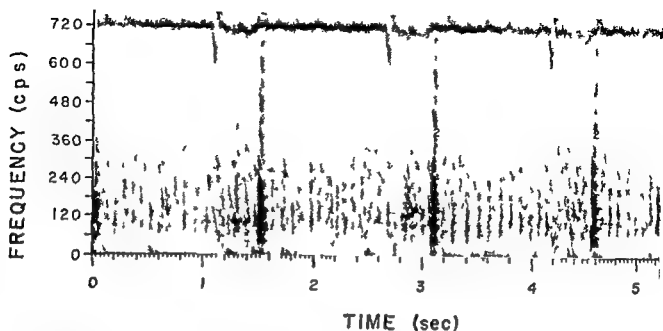


FIG 54 Muscle tremor

Repetitive transients due to muscle contractions are demonstrated in the recording from the aortic area. Two factors may result in such an artifact: (1) Straining by the patient in holding his breath; (2) shivering, often without the knowledge of the patient or physician from cold room.

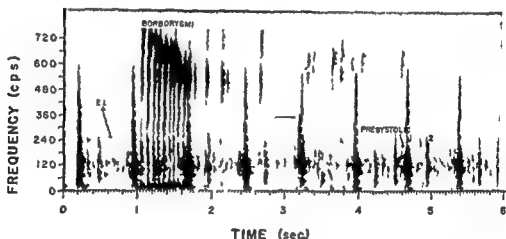


FIG 55 Artifacts in recording in apex in M. M. (242664) 38 year old patient with mitral stenosis. There are borborygmi—a series of rather musical transients—and electrical interference at 240 cycles. The sharp first sound and pre systolic murmur are evident.

fering body noises. The breath sounds are more troublesome in the aortic and pulmonary areas borborygmi at the apex. Muscle noises are mainly the result of shivering. The patient may himself not be aware of chilliness—insensible shivering. The room in which recording is done should be kept warmer than most rooms—certainly 70° F or higher.

In the oscillogram all artifacts are vibrations like cardiovascular sound. They may be difficult to differentiate from meaningful information of

cardiovascular origin since the only means one has for identifying their artifactual nature is the lack of constant relationship to some element of the electrocardiogram and the failure to recur with each cardiac cycle. On the other hand the body noises, as well as many of the major categories of ambient noises have spectrographic characteristics which permit their identification in the SPCG.

The spectrographic characteristics of the breath sounds are described in a later section (p. 487 ff).

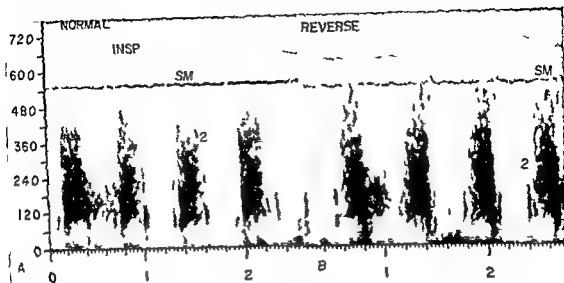


FIG. 4. A filter artifact.

The recordings are from the apex of a patient with rheumatic mitral regurgitation. When analyzed in the customary fashion the beginning of the murmur (1) was seen to have a suspiciously sharp front. It was expected that a three-fold characteristic of the filter system was responsible for such sharp front. The same segment of sound was played back in reverse into the analyzer. It was then seen that the sharp front was indeed largely artifact and that some detail was destroyed when the recording was made in the usual manner. This artifact fortunately not too pronounced in the spectral phonocardiograph of current design should be eliminated insofar as possible in future designs.

Borborygmi (Fig. 5) occur as a series of transients which often have a rather musical quality. They may look rather like tympanic percussion note (p. 47) the similarity is probably no accident. Muscle sound (Fig. 6) likewise present as a series of closely spaced transients. In children speech or other sound of laryngeal origin may be a problem but in the case of ambient speech sound is readily identified in the spectrogram by its harmonic pattern. The noise of moving rarely a problem likewise displays harmonic pattern.

ANALYSIS. The application of the microphone to the chest with air seal attenuates ambient noise by as much as 20 db. However the phonocardiographer quickly becomes aware of the fact that the noise level in most laboratories is appreciably higher than one might at first guess. The ear quickly learns to recognize sound that is not pertinent. The oscillographic phonocardiograph cannot do that. Telephone bell and speech have a characteristic spectrographic appearance in the normal noise. Electrical interference is the major difficulty. Again this is more easily identified in the spectrogram since 60 cycle interference for example is seen as a line at 60 cycles

ARTIFACTS UNIQUE TO SPECTRAL PHONOCARDIOGRAPHY. The sharp fronts the introduction or rather display of high frequency component not truly there when the filter system is hit hard. False splitting of transient again when the filter system is overloaded—these artifacts unique to spectral phonocardiography are illustrated and discussed in Figures 76 to 83 and also in the Technical Appendix (p. 499).

In the oscillographic phonocardiogram time is on the horizontal axis and overall intensity on the vertical axis. Predominant frequency is roughly indicated by the spacing of the oscillations; the more closely spaced are the vibrations the higher is the dominant frequency. In the spectral phonocardiogram time is the horizontal axis as in most physiologic recordings but the vertical axis (ordinate) is frequency scale in cycles per second usually in a span from 0 to 750 cps. Intensity at various frequency levels is indicated by degree of blackness (density) in the given portion of the recording.

PCG is the abbreviation which will be used at times for phonocardiogram or phonocardiography or phonocardiograph (the context will make clear

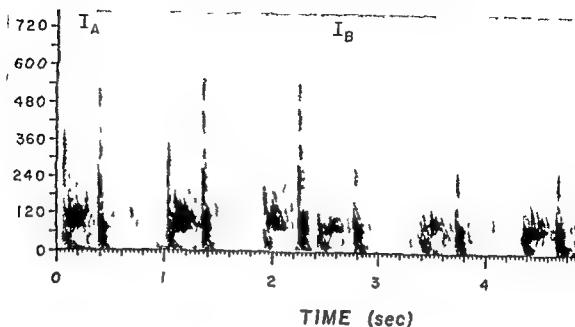


Fig. 57

Fig. 57 Another artifact of the filter system

The experiment repeated by the *c* recordings was suggested by suspicions that artifactual high frequency components are added by the filter system in the case of sharp sounds of high intensity. Recording A in each pair of records was made in the usual manner. Recording B was made after passing the sound through a pass band filter which cut off components above 100 cycles. For analysis II and III the sound was put onto the magnetic recording disk of the analyzer at the same level of amplification but was played back during the analysis itself at three grades of amplification increasing from I to III. For analysis IV, V, and VI the sound was put through the analyzing filter system with the same amplification but in recording, onto the magnetic disk of the analyzer three grades of amplification were used increasing from IV to VI. (The sounds are from the pulmonary artery of a 15 year old boy with active rheumatic fever.)

The records demonstrate that regardless of the stage at which additional amplification is applied, the analyzer displays frequencies appreciably above the level of cutoff of the pass band filter when the sound is very loud. Whether the components of higher frequency displayed are truly artifactual depends on whether the pass band filter, which of course cannot be expected to cut off completely abruptly at 100 cycles, is likely to pass information up to 600 cycles for instance. In recording IIIB such is in fact impossible with the pass band filter employed which produces in attenuation of 21 db per octave. This is then a filter artifact, but one which is not pronounced if overloading is avoided.

which is meant) and *SPCG* for the spectral equivalent. Sometimes *oscillogram* will be used for the conventional *PCG*, and *spectrogram* for the *SPCG*.

The spectral phonocardiogram provides a visible representation of the same information which the ear derives from sound. There are theoretical reasons to anticipate superiority of the visual method for the study of cardiovascular sound. In evolution hearing developed later than vision (744). Vision is superior to audition in many respects. Pleasure information and protection are more dependent on vision. Incidentally as indicated earlier, the two do not blend well. Sight

competes seriously with hearing, and usually takes the upper hand when the two senses operate simultaneously. These facts are related to the observations that blind persons are good musicians and that one closes his eyes to enjoy music to the fullest extent.

The *SPCG* closely resembles the mental image of heart sounds and murmurs. Potentially at least as much information can be displayed as can be made out by the auditory mechanism, still more can be displayed since the *SPCG* suffers from no physiologic masking, no fatigue phenomena, and no peculiarities of intensity response at various frequency levels, and since it has a time

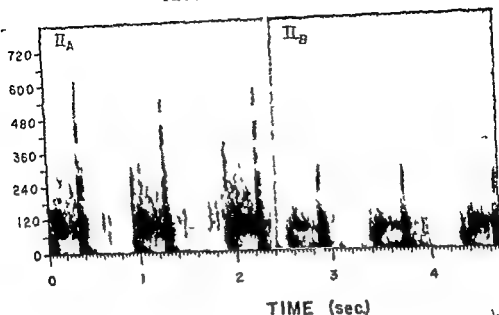


FIG. 57B

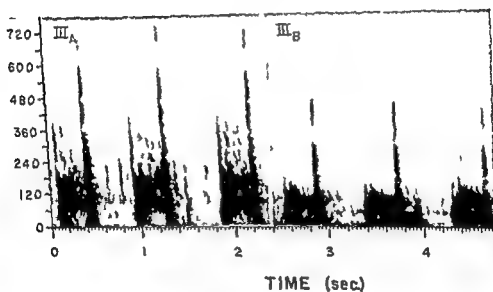


FIG. 57C

resolution superior to that of the ear. The improved resolution of time results from the spreading out of the frequency spectrum even if plotting is not discernible at points of higher intensity. It is likely to be evident in portions of the frequency scale where the plot components are less intense. The spreading out of the frequency spectrum

permits a display of the full dynamic range of cardiovascular sound; for example, a very faint diastolic murmur can be accurately represented in its true intensity proportions in the presence of a very loud systolic murmur (Fig. 59).

Since it emulates the ear, spectral phonocardiography is ideal for teaching clinical auscultation.

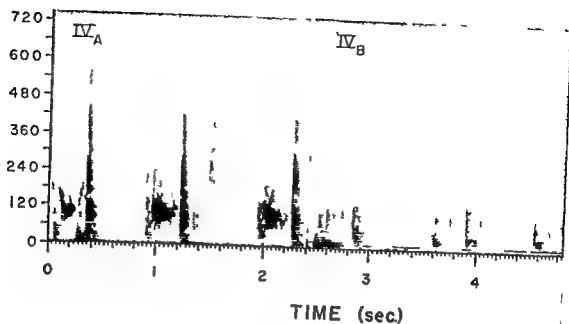


FIG 57D

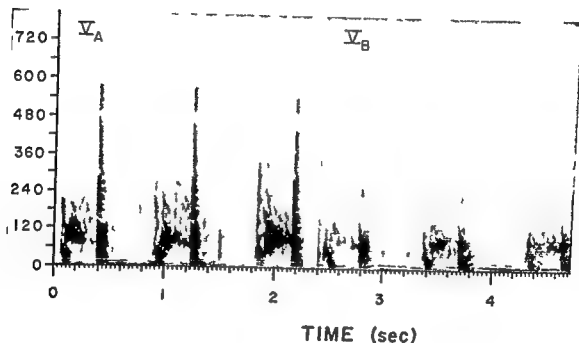


FIG 57F

for detailed presentation of medical sounds on the printed page and for precise quantifiable recording of the course of certain forms of heart disease and the effects of some types of cardiovascular surgery.

Why do phonocardiography (1429)? The reasons are at least three.

1. Phonocardiography has as one of its main

functions to teach our ears what they should be hearing with the aid of the stethoscope. It encourages self-criticism during auscultation (442).

2. Phonocardiography provides valuable documentation particularly in the case of valvular heart disease bearing on the course of disease and the effect of cardiac surgery. It keeps the clinician honest. Dr. William Dock makes the following

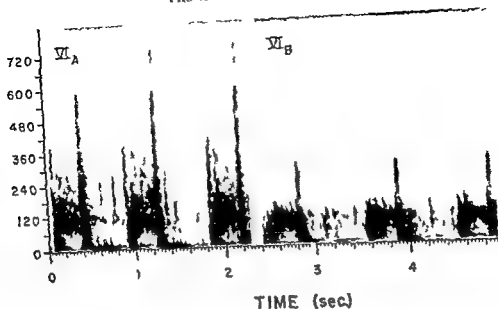


FIG 57

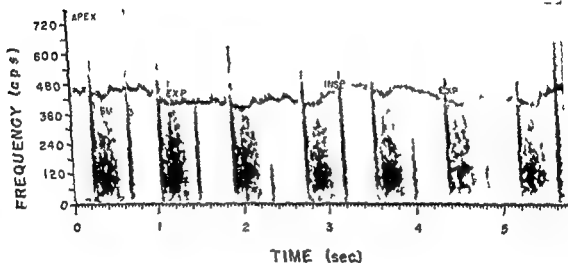


FIG 58 Microphone artifact from excessively intense apex impulse

J.Y. (6364) age 16 years has rheumatic mitral regurgitation with moderate pulmonary hypertension by cardiac catheterization. A forceful impulse was felt at the time of the first heart sound and the third sound. The mapping sound recorded immediately over the apex but at probably are artifactual in their appearance owing to overloading of microphone amplifier. The filter system of the spectrograph also cannot be completely exonerated (see Fig. 51) (see Fig. 171 for split S in this patient).

statement. One should no more think of sending a patient for valve surgery without a phonocardiogram than one would in the old days think of sending a patient to Trudeau without a chest X-ray (see page 100 for a similar quotation from the writing of Dock.) Cardiac surgery—

vascular surgery, closure of septal defect, closure or excision of a ductus arteriosus, etc.—should be followed by pre- and post-operative recording. The evolution of heart disease, especially of valvular and congenital type, is well followed by phonocardiography. The following is another



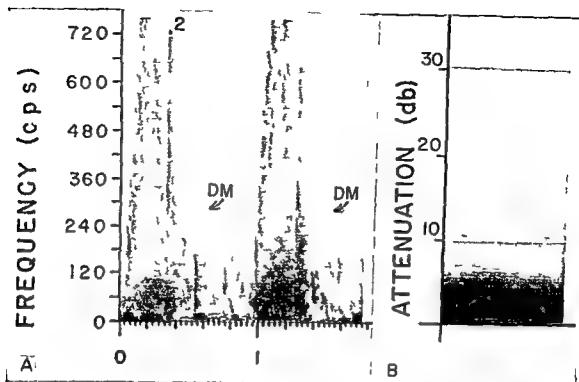


FIG. 59 Display of large dynamic range in SPCG

In this case of rheumatic aortic stenosis and regurgitation the typical Christmas tree pattern of the murmur of aortic stenosis is displayed as well as the decrescendo pattern of the very faint diastolic murmur. In the display of the wide dynamic range of cardiovascular sound spectral phonocardiography improves greatly on the performance of conventional oscillographic phonocardiography. In the example given here (1) the systolic murmur is estimated to be about 60 db more intense than the diastolic murmur a 1000 fold difference. There is no way by oscillography that the two murmurs can be simultaneously displayed in their true proportions. In order to record the diastolic murmur with a maximum height of 3.0 mm the systolic murmur would have a height of approximately ten feet. Using pass band filters will not solve the problem since the tremendous dynamic range is present at the frequency level where the diastolic murmur is most intense (about 280 cps in this case). Use of voltage limitation introduces distortion. In its ability to encompass the large dynamic range of sound spectral phonocardiography resembles the ear. Technical developments in spectral phonocardiography must take this dynamic range into account. In the type recording maximum dynamic characteristics must be provided and the display medium must permit wide possible density grading from black to white. In this regard photographic display has a considerable advantage over the direct written record. The display on electro-sensitive paper provides a dynamic range of about 15 db (2). This deficiency accounts for the homogeneous blackness of the lower frequency range of the systolic murmur in 1. The harmonic pattern of this portion of the murmur is completely obscured.

quotation from Dock (3) (7 p. 64)

The permanent objective record supplied by phonocardiograms are as valuable in managing heart disease as blood smears in leukemia, chest films in pulmonary tuberculosis or electrocardiograms in coronary disease. Perhaps their greatest value is in teaching us to be better doctors at the bedside and in the office and by making us less prone to error when the phonocardiogram is not available.

3 The phonocardiogram can provide information not available by ear for example (a) precise

time measurements as in the case of the delayed first sound in mitral stenosis and the interval between the second sound and opening snap—measurements of quantitative significance in mitral stenosis (b) demonstration of low frequency sounds which are beyond the perception of the ear.

And of course the ability to study the record at leisure and again and again is a considerable advantage. Ability to represent the auditory impression on the printed page is an aid to education

and to scientific progress. Phonocardiography is especially useful when it is elevated from the domain of mere observation and used (1) for making semi quantitative physiologic estimates, as of the level of left atrial pressure in mitral stenosis from the S<sub>1</sub>OS interval (p. 292) and of right ventricular pressure in pure pulmonary stenosis by the interval between the aortic and pulmonary components of the second sound (p. 382) and (2) in conjunction with physiologic and pharmacologic maneuvers designed to modify the heart sound (see Chapter 22).

The expense of phonocardiographic equipment

and the complicated nature of the technique might make it seem doubtful that general clinical use will be forthcoming. However it is not improbable that phonocardiography, specifically spectral phonocardiography (which will in my opinion become the method of choice) is now in a position comparable to that of electrocardiography about 1910. At that time there was expressed serious doubt that electrocardiography could ever prove clinically practicable. It is not inconceivable that the cumbersome research tool can be streamlined for clinical use just as was the electrocardiograph.

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## CHAPTER 8

# *The Structure and Function of the Heart Valves*

The heart valves are intimately related to the fibrous skeleton of the heart which is also the origin and insertion for the spiral myocardial bundles. The relationship of the four valves to the fibrous skeleton and to each other is demonstrated in Figure III 19 of reference 580.

The external topography of the heart valves was shown in Figure 37. In Figure 60 to 64 are presented radiographic demonstrations of the valve locations.

**SEMILUNAR VALVES** (Fig. 65) Since the mechanics of the semilunar (arterial sigmoid) valves, aortic and pulmonary, is simpler than that of the atrioventricular (venous) valves, tricuspid and mitral, these will be discussed first. The cusps of the semilunar valves, normally three in number, are membranous sacs which in the position of closure fit snugly against each other providing mutual support. Each cusp has a thinner, the lunula, in which only minimal structural strength is present and necessary because of this mutual support. (Penetrations occur most often, perhaps, in the lunulae (1031).) A fibrocartilaginous nodule, the corpus aratum, is located in the center of the margin of each cusp. Each of these nodules is so shaped that together the three fit to form a sphere.

Beyond the cusps the vessel is dilated into the sinuses of Valvula. This feature has the effect of insuring that the cusps do not become plastered against the wall of the aortic root during ventricular ejection, a circumstance which would render subsequent closure of the valve difficult and might, furthermore, interfere with coronary flow.

There is confusion in the nomenclature of the heart valves (781). The one followed here—cusps

the most easily remembered—the anteriorly located pulmonary valve has anterior, right and left cusps, the posteriorly located aortic valve has right coronary, left coronary, and posterior non coronary cusps (see Fig. 66).

The opening of the semilunar valves is a simple matter of the pressure in the ventricle exceeding that in the aorta, is the result of ventricular contraction. Cinephotographs of the function of post mortem aortic or pulmonary valves, with ventricular systole simulated by a pump, provide an accurate picture, since there is little evidence that anything but hemodynamic factors, reproducible in the model, are involved. Such studies indicate that the valves are not completely open during systole but rather maintain an intermediate position. This tendency for a flap to be drawn toward the central axis of the stream of flow can be demonstrated by blowing across a sheet of paper which is held by its proximal edge (Fig. 67). The distal part of the paper which hangs down will be elevated, or if the paper is lying completely free, one may be able to raise it by a jet of air blown across it. Bernoulli's principle is doubtless operating in these situations, including that of the heart valves.

As is known to Leonardo, the aortic orifice is triangular (cf. Fig. 68) when the valve is open, is a result of the intermediate position of each cusp. Quain (1236) gives the cross-sectional area of the base of the aorta as about 23 sq cm, and McMillan (1088) estimates the area of the triangular orifice as 26–35 sq cm.

Closure of the heart valves occurs with virtually no regurgitation—for practical purposes none. For example, in normal subjects no regurgitation into the left ventricle can be demon-



FIG 60 Anticardiographic demonstration of the location of the tricuspid valve (TV) (Courtesy of Cooley and Sloan (1961))

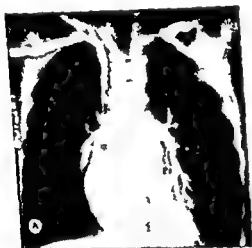
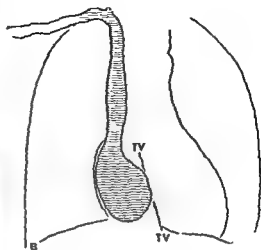
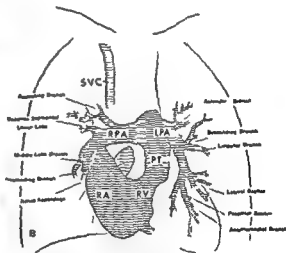


FIG 62 Angiocardiographic demonstration of location of pulmonary valve (between RV right ventricle and PT main pulmonary artery). Bulging of the pulmonary sinuses of Atrial valve is demonstrated (Courtesy of Cooley and Sloan (1961))



trated on aortography. A pulling in of the cusps in the wake of the ejection jet placed the cusp in an optimum position for efficient closure (670). The Leonardo da Vinci theory (see p. 36) that vortices form on the outer aspect of the cusps in the sinuses of Atrial valve and that when ventricular ejection flow ended the jet uncoiled like tightly wound watch springs and pushed the cusps but remained unlikely although recently in 1927 Hochstein (193) proposed this view. Ceradini, Luciani, Henderson and others (see p. 38) were

of the opinion that breaking of the jet was the most important factor in closure of the arterial valve. The ejection stream was visualized a column of blood whose momentum carried it on briefly even after contraction of the ventricle ceased. The arterial cusps were thought to be drawn in in the wake of the jet (See Fig. 69).

The mechanics of bicuspid and quadricuspid valves (see p. 37 for some historical details) and the phenomenon of trigonoidization may be discussed at this point especially since these are

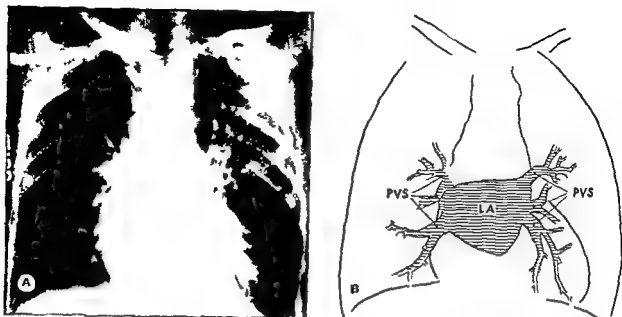


FIG. 62 Angiocardiographic demonstration of the location of the mitral valve

The mitral ring is represented by the somewhat flattened left contour of the left atrium (Courtesy of Cooley and Sloan (291) )



FIG. 63 Rheumatic aortic stenosis with calcification (arrows) of the aortic valve

This 23 year old male had syncope attacks, one of which was fatal. The heart shows surprisingly little enlargement. *A* Conventional film. *B* Heavy exposure at one sixtieth second to improve demonstration of the aortic valve calcification visible by fluoroscopy. Autopsy confirmed the radiologic diagnosis of aortic valve calcification (Courtesy of Cooley and Sloan (291) )

biological variants of the normal situation and perhaps cannot be considered strictly abnormal. The bicuspid aortic valve cannot open efficiently and tends to produce some degree of obstacle to forward flow through the orifice. The fact that slight regurgitation often develops at bicuspid valves suggests that closure in these valves may

not be as reliable as in the tricuspid valve however it is more likely that the regurgitation is the result of the secondary atherosclerotic change to which these improperly engineered valves are prone and of the hypertension or coarctation of the aorta with which bicuspid aortic valve is often associated.

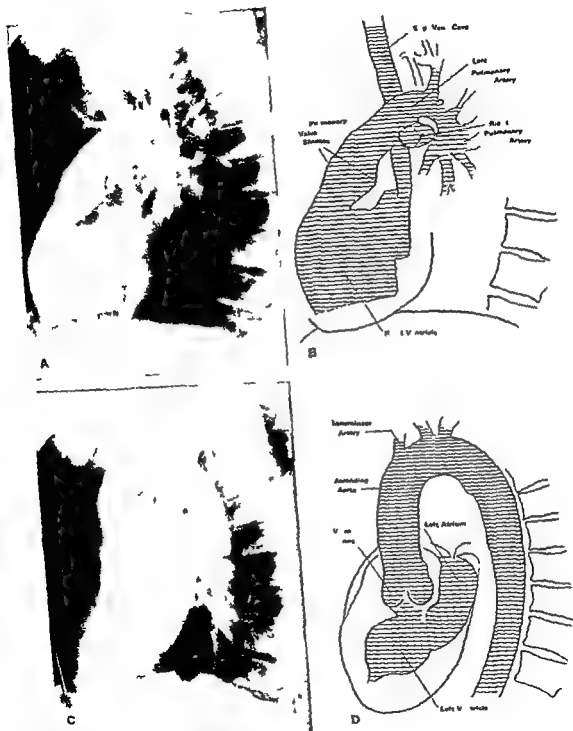


FIG. III. Angiocardiographic demonstration of the location of the pulmonary (A and B) and aortic (C and D) valves. Lateral views. (Courtesy of Cooley and Sloan (1961).)

Quadruped valves have an unfavorable ratio between the apex base dimension and the base dimension. Where the bicuspid valve functions better than optimally in opening, a quadruped

valve is relatively weak in performing the function of guarding the mouth of the great artery during ventricular diastole.

Quadruped valves occur more commonly at



FIG. 6. Functioning heart valves

In *a* and *b* are presented successive frames from a motion picture (made at the speed of 24 frames per second) of the closing of the pulmonary valve in an isolated surviving beef heart. In *c* to *f* are presented comparable series in the closure of the tricuspid valve in the same heart. The tricuspid valve took about twice as long to close. The tricuspid valve closed first at the middle. Faster closure of the arterial valves may be in part responsible for the fact that the second heart sound is usually shorter, with more high frequency components than the first. (From film I MF 5162 made by the Armed Forces Institute of Pathology.)

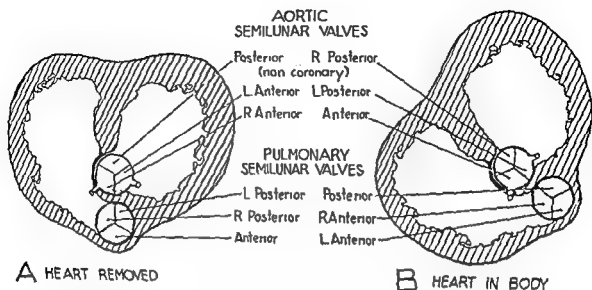


FIG 66 The nomenclature of the aortic and pulmonary cuys (from Houlden 1905) The system indicated in the left hand column is the one which has been used in this monograph



FIG 67 Blowing across a paper provide a graphic classroom demonstration of why the arterial leaflets are held in a mid position during systolic ejection

the pulmonary orifice. A quadricuspid pulmonary valve has been found in an incidence between about 0.01 and 0.25 per cent in a series of properly studied autopsies (798). Occasionally (798) pulmonary regurgitation occurs with it as was predicted by Leonardo da Vinci; that it does not occur more often is probably related to the relatively low pressure in the pulmonary artery under normal circumstances.

Bicuspid state occurs more often at the aortic

valve. Of all patients with coarctation of the aorta 20 to 40 per cent have bicuspid aortic valve. The valves commonly undergo calcific athero sclerotic and even stenotic change and even more commonly display regurgitation in some degree. Rheumatic fever may cause fusion of two aortic cusps with resulting simulation of a congenitally bicuspid valve.

Chalmers (292) pointed out that what he dubbed trigonoidization, likely to occur in the case of the thin walled pulmonary artery especially under condition of high pulmonary flow. The illustration (Figure 70) will aid in understanding the phenomenon. The result is that during ejection of the right ventricle the pulmonary cusps are pulled into a sector of the valve orifice. The portion of the free margin of the cusp stretched in this manner may function as a non generator possibly being related particularly to the muscular variety of so called functional murmur (see p 244). In connection with the concept of trigonoidization the following observations are of interest and possible pertinence when considered together. (1) With a properly constructive pick up device it is not a systolic murmur can be demonstrated at the orifice of the chest in all persons (610). (2) Intracardiac phonocardiograms demonstrate a systolic murmur in the pulmonary artery in all cases (896). (3)



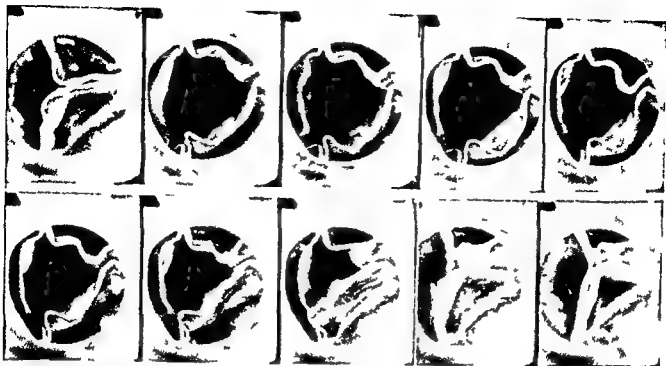


FIG 68 A cycle of normal aortic valve movements showing the opening and subsequent closure. Successive cinematographic frames made with simulation of ventricular systole in a normal human heart by means of a pump. Courtesy of McMillan (1958) and the British Heart Journal.)

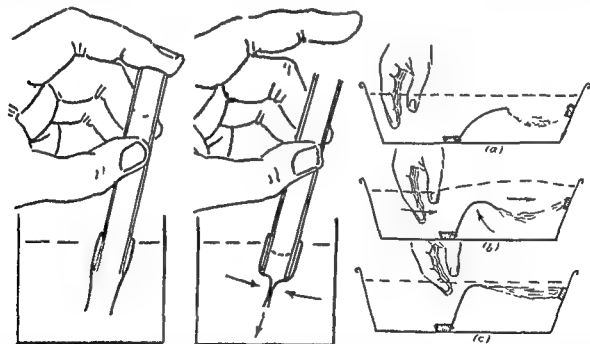


FIG 69 Demonstrations of the wake of the jet phenomenon. (Left) Compare the situation schematized here with the atrioventricular valves or the arterial valve. The full of fluid in the tube is analogous to the propulsion of blood into the ventricle by atrial systole or into the aorta by ventricular systole. Because of momentum the fluid fills below the general level of the fluid in the vessel and the flaps, which are analogous to the valve cusps, come together. (Right) In the wake of a wave the flap is pulled in toward the center of the stream. (From Henderson and Johnson (1970).)

Phonocardiograms recorded directly from the surface of the pulmonary artery in dogs and man demonstrate a systolic murmur in all cases (1907). (4) When phonocardiograms are taken directly

from the surface of the pulmonary artery in dogs in which the pulmonary valve leaflets have been visualized the systolic murmur disappears (1907).

The question of a role of the ventricular murmur

lature in closure of the arterial valve is ruled mainly in connection with causes of bilateral diastolic murmur in association with ventricular failure (410) or with anemia and dilated ventricle. In brief the evidence is such that an explanation for such a murmur must be sought elsewhere than in a dilatation and relaxation or weak contraction of the muscular ring surrounding the aortic and pulmonary orifices. In the case of the aortic orifice there is a separate myocardial bundle the deep bulbo-papillary (1002 1028 1281) which surround the outflow tract. This muscle contracts of course during ventricular systole and probably has no persisting tension in early diastole. Therefore imperfect contraction would not be a likely factor in failure of the integrity of the aortic orifice. Furthermore, in atonic and dilated state would probably not be a factor either although it is said that if the deep bulbo-papillary muscle is selectively cut in experimental animal aortic regurgitation occurs immediately. The fact that the aortic valve are water tight to the extent of withstanding very high pressures applied on the aortic side in autopsy heart is further evidence against a muscular element in arterial valve closure. The aortic valve is part of the aorta not the ventricle and is not affected by dilatation of the ventricle.



FIG. 0 Trigonization of the pulmonary valve (above) Schematic representation of successive degrees of dilatation and trigonization (Below) Photograph of the pulmonary orifice in a necropsy specimen which was fixed with a plug of cotton in the conus (From Chalmers '67)

Brock (179B) has assembled phylogenetic anatomical and clinical data suggesting that muscular function is important to the closure of the pulmonary valve. He states that the conclusion that the control of the pulmonary outflow is a purely mechanical function of the valve cups and is not supported by muscular action in the mammalian heart is almost certainly incorrect; it is certainly incomplete. It is plausible to presume that trigonization (which results

in not only a systolic but also a diastolic murmur (330A)) would be more likely to occur if systolic function of the infundibular musculature were defective. Pulmonary regurgitation may persist for some days after open direct surgery for Fallot's tetralogy (179B). Brock (179B) describes a patient who had had open repair of pure infundibular stenosis and showed pulmonary regurgitation only during a subsequent pregnancy.

In discussing the paper of Anders (19) Stewart referred to the existence of a muscle specifically surrounding the outflow tract of the left ventricle and stated that if we expose the heart of an animal and insert a fine curved knife through the wall of the ventricle so as to divide the muscle without injuring in any way the aortic cup, a systolic murmur of aortic insufficiency will at once become audible. There is a great need for a careful and critical repeating of this experiment which I have found described nowhere except in Stewart's discussion taken down photographically. For the reasons stated above a muscular factor in aortic regurgitation is doubtful but the matter cannot be considered settled until Stewart's experiment is carefully repeated.

**THE ATRIOVENTRICULAR VALVES** The mitral valve has two cusps (anterior-septal or aortic-posterior or mural) with chordae tendineae which attach to two papillary muscles. The tricuspid valve which is attached by chordae tendineae to three papillary muscles has two main cusps (largest anterior infundibular, smaller posterior



FIG. 71 The fanning out of the collagen bundles of the chordae tendineae in the sub-tineae of the mitral cusp. That each papillary muscle sends chordae tendineae to both cusps is evident. (From Rushmer (1321))

inferior) and a smallest intermediate (septal) cusp. The mechanics of the two AV valves have many identical features despite the variation in structure. In fact the difference in structure is not so striking since the intermediate cusp of the tricuspid is often so small that the valve is essentially bicuspid and occasionally even the mitral valve may have a small third cusp. When 'mitral

valve' is used in the following discussion assume that both AV valves are meant.

The orientation of the tricuspid inflow tract to the pulmonary outflow tract is such that an angle of about  $60^\circ$  is subtended by the two paths. On the other hand the corresponding mitral and aortic paths on the left side are essentially parallel to each other (383). It has been thought that the

arrangement on the right side is more conducive to regurgitation at the AV valve than that on the left.

The chordae tendineae are collagenous cord fundamentally identical to ordinary tendons as indicated by the name. In the cusps the fibers of a given chorda fan out in the substance of the valve membrane. An attractive fan trier is revealed best on transillumination of the cusp (figure 71) results. A comparison with architectural fan triers such as that in the cathedral at Wells, England has been made (179). A point of possible functional significance is the fact that a given papillary muscle end chordae to two cusps.

Undoubtedly the most important factor in all valve closure is a change in polarity of the pressure differential across the valve orifice. In the case of the atrioventricular valves the rise in ventricular pressure (through contraction of the ventricle) above that in the atrium is the most important factor in closure.

Possible ancillary factors contributing to closure of the AV valves will be discussed in the following order:

1 Contraction of the muscle surrounding the mitral ring reducing the cross-sectional area of the orifice,

2 Contraction of the papillary muscles drawing the cusps together,

3 Pulling in of the cusps in the wake of the jet produced by atrial systole (670)

4 Contraction of atrial musculature extending onto the AV valves.

The Leonardo da Vinci mechanism discussed in connection with the emuln valves is applicable here as there.

The evidence that contraction of the muscular ring may contribute to closure of the mitral orifice is two-fold. Motion pictures of the ventricle and open while still beating revealed shortening of the portion of the ventricle constricting the ring (1407). Motion pictures of the mitral valve of the beating heart photographed from the atrial aspect likewise show reduction of the cross orifice size with ventricular contraction (67). In the second place mitral regurgitation is accompanied calcification of the annulus fibrous mitral even when the cusp and part of the

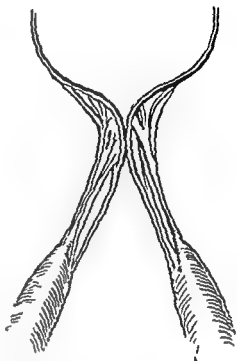


FIG. 72 As is demonstrated in this simple sketch the atrioventricular valves probably appose over an appreciable portion of the margin. This provides a certain margin of safety, e.g. the cusps are sufficiently large that mitral regurgitation will not occur with slight or only moderate ventricular distension.

valve mechanism in its normal. It has been suggested (1391) that the contraction of the muscular ring is frustrated in this condition. However this situation is not entirely clarified. Together the AV valve cusps have a surface area which exceeds the cross-section area of the corresponding orifices (Fig. 72). Probably the cusps will suffice to close the orifice even when the ventricle is considerably dilated. The anterior or aortic leaflet of the mitral valve can compensate for considerable deficiency of the posterior leaflet with the result that mitral regurgitation is unlikely to occur from lesions limited to the posterior cusp.

Apposition of the cusps through contraction of the papillary muscles which end chordae to opposing cusp is inferred (1321) from the anatomy (Fig. 71). Each papillary muscle is connected by chordae to both leaves of the mitral valve, for example. The papillary muscles are thought to contract early because of their endocardial location. Contraction of the papillary muscles, which

ever role it may have in the closure of the atrioventricular valve, is important in preventing partial eversion of the cusps into the atrium with ventricular systole and regurgitation is a result thereof. Mitral regurgitation may develop from improper function of the papillary muscle, for example from infarction subsequent to coronary occlusion, without actual rupture of the papillary muscle or of the chordae. When the ventricle is acutely dilated efficient closure of the AV valve may be impossible because the papillary muscle and chordae combination is too short. With marked dilation of the ventricle the papillary muscles may become "absorbed" in the rest of the myocardium, that is, they are so stretched that they lose their separate identity.

In the closure of the arterial valves the "wake of the jet" mechanism is thought to be an important factor. That a similar mechanism may be operating in the case of the AV valves, with atrial systole being responsible for the jet, is suggested by several observations and experiments.

1 In dogs with sinus rhythm, injection of dye into the left ventricle reveals little or no evidence of mitral regurgitation. Appreciable regurgitation can be demonstrated if atrial fibrillation is induced (325). Henderson and Johnson (670) were inspired to investigate the wake of the jet mechanism by observing the remarkable ability of the heart valves to close without attendant regurgitation.

2 The length of the PR interval is one of the main factors in determining the intensity of the first heart sound. The degree of separation of the cusps of the AV valves at the onset of contraction of the ventricles is seemingly the immediate factor largely determining the intensity of the first sound. These considerations are pointed up by the chart presented in Figure 129. In a dog with surgically produced complete atrioventricular dissociation the intensity of the first sound was plotted against the length of the preceding PR interval. When the atrium contracts before the ventricle by an interval corresponding to a PR interval in the normal range the first sound shows minimal intensity. This is interpreted as indicating that the cusps are relatively close together and the assumed close position of the

cusps can in turn be interpreted as evidence that they have been drawn together in the wake of the jet produced by the just preceding atrial systole. With shorter PR intervals than normal it is thought that the cusps are widely spread, and with longer ones than normal it is thought that the cusps have again had an opportunity to resume a neutral position after having been drawn in in the wake of the preceding atrial jet.

In man, a faint first heart sound occurs with prolongation of atrioventricular conduction, as in acute rheumatic fever. A variable first heart sound occurs with the Wenckebach type of second degree heart block and particularly with complete heart block. *Brut de canon* was a term the French gave to the occasional booming first heart sound that occurred with complete atrioventricular dissociation.

3 There are suggestions from the right atrial pressure curves (832) and jugular venous pulses (435), in cases of complete heart block, that closure of the tricuspid valve occurs momentarily following atrial systole.

The above observations, which seem so well accounted for on the basis of the experiments of Henderson and Johnson (670), are difficult to reconcile with the report of Rushmer and colleagues (1326) that in the intact dog radiopaque markers placed on the mitral valve show little movement during the cardiac cycle. Current thinking relates the heart sounds more to a tensing of the belly of the valve cusps than to a collision of cusp margins. Is it not possible that Rushmer has studied mainly motion of the cusp margin and that considerable motion of the cusp belly with its rolling in the wake of the jet may occur independent of movement of the margin of the cusp?

In animals atrial myocardial fibers have been shown to extend into the AV valves. There are less striking in man than in lower animals. It is doubtful that they are of any functional significance in man at least. Ehringer (432) described the beating of this musculature for some time in an excised animal heart and thought it might account for the partial closure of the AV valves in pre-systole as observed by Deming.

It will be clear from the above discussion that

it is impossible to reproduce the function of the AV valve in the necropsy heart in as complete detail as one can with the arterial valves. Although useful information can be gained from cinematographic studies of normal and diseased mitral valve in a pump model the artificiality of the set up must be constantly borne in mind.

As pointed out by Talbot (1958) an important function of both the AV and the arterial valves may be to increase the efficiency of blood transfer. Both sets of valves funnel blood into the ven-

tricle or artery and tend to prevent the turbulent break up of the blood stream—a development which would lead to less efficient flow and to murmur production.

The ultrasonic Doppler method for demonstrating valvular function (1959A) and the correlations with cardiovascular sound promises to provide valuable information. It appears for example that opening of the normal AV valve is indicated in these recordings and also the movement accompanying atrial systole. Direct observation of these movements in the normal heart has hitherto been impossible.

## CHAPTER 9

# Temporal Relationship between Heart Sounds and Other Cardiovascular Phenomena

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Basic to remembering the temporal relationships of the first and second heart sounds are the premises, now reasonably well established, that the first and second sounds are valve closure noises and that valve closure occurs, probably with some very slight lag when there is a change in the polarity of the pressure differential across the valve orifice. Therefore, in simultaneous recordings of pressure on the two sides of the valve, the valve will open or close when the lines representing the respective pressures cross each other. As a generalization, too, it can be stated that valve opening is silent (see p. 12) for a discussion of this mooted matter). And it must of course be kept in mind that the first sound is a composite noise associated with closure of the two AV valves and the second sound a composite of the closure sounds of the two arterial valves.

The chart in Figure 73 presents information derived from cardiac catheterization and other sources. Closure of the aortic or pulmonary valve and the part of the second heart sound produced thereby corresponds to the mesurium in the arterial pressure pulse of the corresponding great vessel. The closure of the AV valves and the resultant first heart sound occurs when the curve for ventricular pressure crosses that for pressure in the atrium.

When AV valve opening is productive of an opening snap as in mitral stenosis this sound occurs at the time that the ventricular pressure falls below atrial pressure. When the arterial valve opening is productive of an opening snap as in

pulmonic stenosis, the sound occurs at the time that ventricular pressure exceeds pulmonary arterial pressure.

The temporal relationship of events in the two ventricles and the effect this has on the relationship of the sounds generated on the two sides of the heart should be considered next. Again, cardiac catheterization has probably provided the most dependable information (166) (169). This information is reviewed in Figure 74. Contraction begins earlier in the left ventricle than in the right with the result that the mitral valve tends to close slightly in advance of the tricuspid. Because of higher diastolic pressure in the aorta than in the pulmonary artery isometric contraction is longer in the left ventricle than in the right. Ejection from the right ventricle begins before ejection from the left ventricle.

Normal asynchronism in the contraction of the two ventricles has been demonstrated in the past in both animals (770) and man (1576). In the last decade electrokymography has been applied to the problem. Comparing the onset of the major ascending limb of the carotid pulse with the corresponding point on the pulmonary artery electrokymogram, Illman and associates (423) in 68 normal young adults found that ejection from the left ventricle began first in 33 subjects by 0.01-0.03 sec from the right ventricle first in 21 subjects by a comparable interval and from both ventricles synchronously in the remaining 14 subjects. On the other hand, using a similar method Lunsford and Iverscher (984) found right ventricular ejection had precedence in all of 8

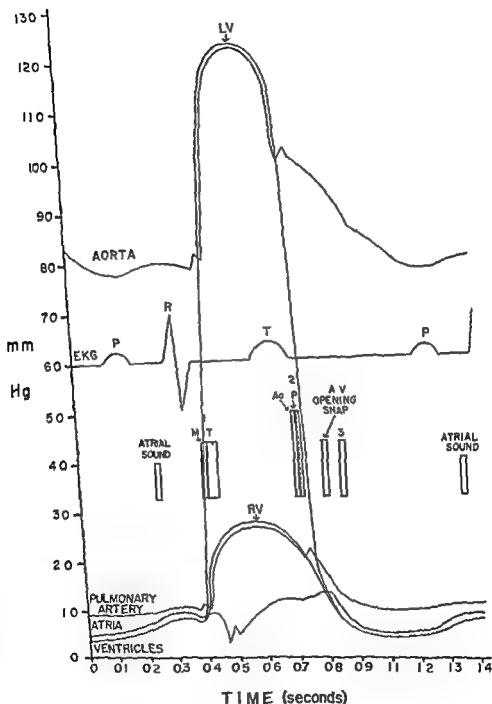


FIG. 7. Temporal relationship of the heart sounds.

Shown are pressure curves in the aorta, left ventricle (LV), right ventricle (RV), pulmonary artery, and atria (which for convenience are assumed to have identical curves of intracavitary pressure) and EKG. M, T, Ao, and P refer to the closure of mitral, tricuspid, aortic, and pulmonic valves respectively. The intervals indicated by the time scale are not intended to be precise. A probable contribution to S<sub>2</sub> produced when pressure in the left ventricle exceeds total pressure in the atria (and the aortic valve opens) is not indicated.



normal subjects by 0.025 to 0.03 sec. Such demonstrations of asynchronism of the onset of ejection cannot be translated directly to time of closure of the atrioventricular valves. Because of

the higher diastolic pressure in the aorta than in the pulmonary artery isometric contraction (after closure of the AV valves) might be expected to be longer in the left ventricle than in the right. By such a mechanism it is possible that mitral closure might slightly precede tricuspid closure and still onset of ejection from the left ventricle would occur later than that from the right. Some studies indeed suggest that isometric contraction is longer in the left ventricle. (On a rational basis, precedence of tricuspid closure over mitral closure might be anticipated since the right atrium contracts before the left, and by the time the ventricles contract the tricuspid leaflets have had time to reach a slightly more closed position than have the mitral leaflets.)

Classically the cardiac cycle is variously divided into phases (1322) which are assigned different names according to the several nomenclatures. The following is an eclectic classification.

*Iso(tolu)metric contraction phase*—between closure of AV valve and opening of arterial. This interval has pertinence in connection with cardiac sound when there is an arterial opening snap, as in pulmonary stenosis or a systolic murmur generated at the arterial orifice because the interval between the first sound and the opening snap or beginning of the murmur is a measure of isometric contraction.

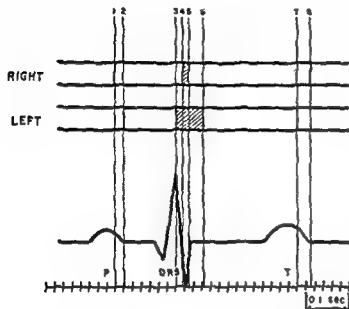


FIG. 74A Diagrammatic representation of the average timing of electrical and mechanical events on both sides of the heart in normal subjects: (1) onset of right atrial contraction (2) onset of left atrial contraction (3) onset of left ventricular contraction (4) onset of right ventricular contraction (5) onset of right ventricular ejection (6) onset of left ventricular ejection (7) end of left ventricular ejection (8) end of right ventricular ejection

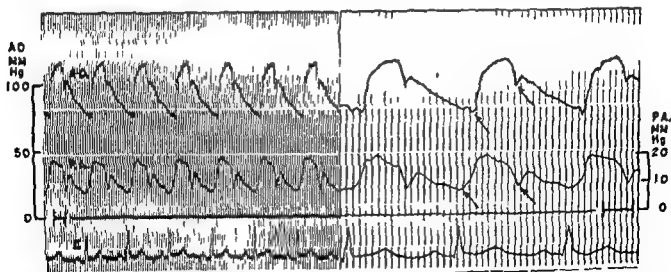


FIG. 74B Simultaneous aortic (AO) and pulmonary artery (PA) pressures in a subject with a normal circulation (illustrating the type of curves from which the information shown in A is derived). Note difference in time between corresponding events in the two curves. Paper speed 25 mm/sec on left (5 mm/sec on right). Time lines 0.04 sec apart. Arrows indicate beginning of ejection and end of ventricular systole. (Courtesy of Braunwald, E. J. and Cornin, (166) and Circulation Research)

*Systolic ejection phase*—which on the basis of aortic pressure and/or ventricular volume curves is sometimes divided into phases of maximum ejection and reduced ejection (Theoretically at least there must be between the time that the ventricle begins to relax and the time of closure of the arterial valves a very brief period which is legitimately considered part of diastole and is usually referred to as the protodiastolic period by Wiggers (1951). It is preferable to avoid this term because of the obvious possibility of a false association with protodiastolic gallop which occurs at quite a different part of the cardiac cycle.)

*Iso-metric relaxation phase*—between closure of the aortic valve and opening of the AV valve. This phase corresponds to the interval between the second sound and a mitral or tricuspid snap if present.

*Rapid filling phase*

*Reduced filling phase*

*Atrial systolic phase*

It will be noted that there is no chronically described phase for the interval between the onset of contraction in the ventricle and closure of the AV valve. *Pre-isometric contraction phase* is a satisfactory designation (JOS 1139). This interval is important in mitral (or tricuspid) stenosis. It has been referred to by Kelly as the *mechanical lag period* the interval between onset of the QRS of the electrocardiogram and onset of ventricular contraction being the *electrical lag period*.

In making measurements in phonocardiograms the convention is to measure from the beginning of one component to the beginning of another. For example the Q1 interval is that separating the beginning of the QRS from the beginning of the rapid deflection or high frequency components of the first heart sound. Splitting of S is measured in terms of the distance from the beginning of the first (usually aortic) component to the beginning of the second (usually pulmonary) component.

The first sound—at least the portion caused by mitral closure—normally begins after the beginning of the QRS of the electrocardiogram by an interval which is about 80 msec for adults (87a) and 0.03 second for children (1034). Strober et al (1449A) found in 100 subjects ages 2 to 3 years that the Q1 interval varied between 0.02 and 0.06 sec with a mean of 0.04 sec.

TABLE 2

*Phonocardiographic time intervals in children* †

| Interval | N   | $M \pm S(N)$      | SD    |
|----------|-----|-------------------|-------|
| Q1       | 117 | $0.040 \pm 0.001$ | 0.010 |
| II-III   | 88  | $0.130 \pm 0.003$ | 0.021 |
| I-IV     | 64  | $0.123 \pm 0.003$ | 0.021 |
| IV-I     | 61  | $0.073 \pm 0.002$ | 0.023 |

N = Number of cases.  $M \pm S(N)$  = Mean  $\pm$  standard error of the mean. S(N) = Standard deviation.

Children under 14 years equal sex distribution fairly uniform age distribution.

† Modified from Table 19 p 9 of Mannheim (1933).

The closest correlation of those analyzed was with height ( $r = 0.36 \pm 0.11$ ). The coefficient of correlation for weight, QRS duration, age, and heart rate were 0.37, 0.20, 0.12 and -0.22 respectively.

The second sound bears no constant direct relationship to the I wave of the electrocardiogram except insofar as the interval between the first and second sound is normally a measure of mechanical systole and mechanical systole bears a relationship to electrical systole as measured by the QT interval.

Leonard Weiler and Warren (87a) found a Q1 interval (that between onset of QRS and first rapid deflections attributed to mitral valve closure) of 0.03 sec in normal subjects. This figure accords well with that of Braunwald et al (166) who found an average interval of 0.032 sec between the onset of the QRS and the crossing of left ventricular and left atrial pressure curves. This interval is prolonged in mitral stenosis (see p 286) because of elevation of left atrial pressure. Curiously it is also prolonged to some extent (on an average (87a) to about 0.070 sec) in systemic arterial hypertension. See p 427 for a discussion of the basis of this delay.

The interval between the first and second sounds is one of the more usable of the available indices of the duration of mechanical systole (1031). The length of systole varies with the heart rate as indicated in Figures 7a to 77. Increased activity of the sympathetic nervous system (1038) and situations such as thyrotoxicosis or pheochromocytoma in which an effective increase in sympathetic activity is produced by the humoral disorder of the disease cause abbreviation of systole out of

proportion to acceleration of heart rate. The curve for duration of mechanical systole in reference to heart rate is different in women and in men for any given rate, systole is usually shorter in men (962), in all positions (upright or supine). The vagus nerve has only an indirect effect on the duration of ventricular contraction, i.e., through effects on heart rate. Mechanical systole is shorter in hyperolemia and longer in hypolemia, although not as long as electrical systole. Changes in potassium and pH also affect the duration of systole. Cridi (234) found that with hypothermia electrical and mechanical systole are prolonged in a parallel fashion to a value three or four times the normal.

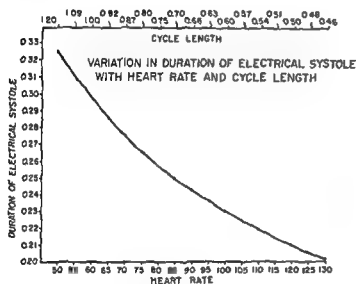


FIG 75 The variation of electrical systole (QT duration) with heart rate (below) and cycle length (above)

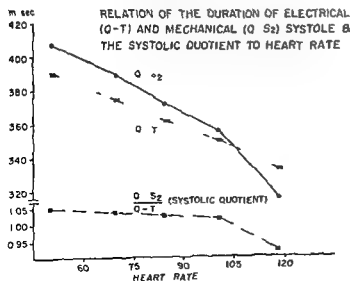


FIG 76 The relation of electrical systole to mechanical systole (From Kubus (923))

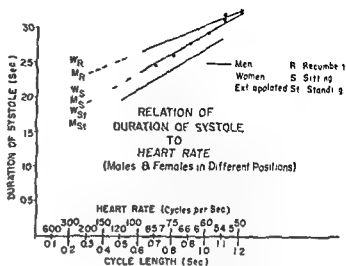


FIG 77 The variation of mechanical systole (from arterial pulse wave) with heart rate and posture. From Lombard and Cope (962). The dashed segments of the line for male subjects were derived by extension of the rest of the line, which is based on actual observations in normal subjects. The data for females was likewise extensive than those for male. The formulae for the straight lines are as follows ( $C$  = cycle length and  $S$  = systolic length in sec):

|                           |                       |
|---------------------------|-----------------------|
| $W_R$ (women recumbent)   | $S = 0.060C + 0.248$  |
| $M_R$ (men recumbent)     | $S = 0.103C + 0.2010$ |
| $W_S$ (women sitting)     | $S = 0.135C + 0.163$  |
| $M_S$ (men sitting)       | $S = 0.147C + 0.145$  |
| $W_{St}$ (women standing) | $S = 0.157C + 0.117$  |
| $M_{St}$ (men standing)   | $S = 0.150C + 0.1212$ |

Table 3 lists some factors responsible for shortening and lengthening of systole. Libraries have been taken in connection with several conditions since information is limited as far as mechanical systole is concerned and is much more ample for electrical systole because of the greater ease of measurement. Partial justification for judiciously using interchangeably information on the duration of mechanical and electrical systole is provided by Blur Wedd and Young (111) who found that the duration of contraction is related linearly to the Q-T interval over a wide range. In 1911 Chier and I (277) found reasonable correspondence of mechanical and electrical systole. The corroborative findings of Kubus (823) in 271 students 20 to 30 years old are presented in Table 4 and Figure 76. Systolic quotient is the term he proposed for the ratio of mechanical systole to electrical systole.

As an exception to the relationship between mechanical and electrical systole Heeklin (661) described a syndrome to which he gave the name

TABLE 3

Factors affecting duration of mechanical systole\*

| Prolongation   | Abbreviation  |
|--|---|
| 1 Female (962)   | 1 Male  |
| 2 Bradycardia  | 2 Tachycardia                                       |
| 3 Increased diastolic volume                               | 3 Reduced diastolic volume e.g. in peritonitis      |
| a In peritonitis (RV)                                      | 4 Thrombotic is                                     |
| b ASD (PV)   | 5 Epinephrine (547 Fig 6) and sympathomimetic drugs |
| c LAD and ASD (LV)   | 6 Hypercalcemia                                     |
| d After compensatory pause or extrastole                   | 7 Anemia  |
| e After saline infusion (104 Fig 6) (Lusada and Barnoff)   | 8 Fever   |
| f Elevation of legs (Weitz and Warneck)                    | 9 Digitalis (204)                                   |
| g In complete AV block when PR interval appropriate (1389) |   |
| 4 Increased resistance to systolic ejection                |   |
| 5 Hypocalcemia   |   |
| 6 Alkalosis  |   |
| 7 Hypothermia (234)  |   |
| 8 Myxedema   |   |
| 9 Quinine quindine atabrine emetine                        |   |

Since there is much more information on the duration of electrical systole than mechanical systole, fibrillations have been taken in preparing this chart. It is not proved in many cases that the same changes are seen in mechanical systole as in electrical systole.

TABLE 4

The relation of mechanical and electrical systole

| Amplitude of systole | Heart Rate | Arterial Pressure | QT (msec) normal | QT (msec) with on gm | Systolic Pressure (mm Hg) | Quinine Quindine Atabrine Emetine |
|----------------------|------------|-------------------|------------------|----------------------|---------------------------|-----------------------------------|
| 50                   | 40-60      | 50                | 418±2            | 388±23               | 100±0.01                  |                                   |
| 115                  | 61-80      | 60                | 413±16           | 344±19               | 104±0.01                  |                                   |
| 60                   | 80-90      | 5                 | 373±18           | 363±16               | 103±0.01                  |                                   |
| 70                   | 91-100     | 16                | 304±11           | 344±20               | 102±0.01                  |                                   |
| 110                  | 106-120    | 115               | 314±18           | 372±12               | 0.96±0.01                 |                                   |

Data of Kuhn

Significance = standard deviation

omissions assigned of energetic-dynamic circuitry in efficiency (Fig 7b). The syndrome is characterized by mechanical systole shorter than electrical systole. (It is interesting that a converse situation occurs normally in the non-fibrillating hedgehog (748) and in the kangaroo (1426) mechanical systole is longer than electrical sys-

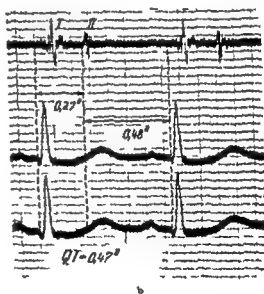
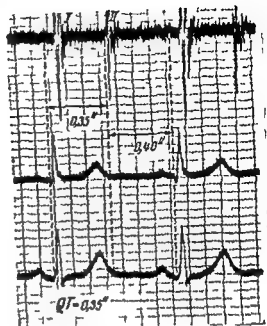


Fig 7. 1 Normal R Heggin's syndrome. Note that mechanical systole is shorter than normal and electrical systole is much longer than normal. (From Heggin (661).)

tole.) Heggin found his syndrome in association with diabetic coma and with liver disease. Kuhn (823) points out that three combinations can lead to the situation of mechanical systole shorter than electrical systole:

- (1) Normal QS, long QT
- (2) short QS, normal QT
- (3) short QS, long QT

The third situation is one to which Hegglin specifically referred as "energetisch dynamische Herzinsuffizienz"

In the electrical systole Bazett suggested the formula

$$K = \frac{QI}{\sqrt{L_3 \text{ etc length (sec)}}}$$

where  $K$  is 0.37 for men and 0.1 for women. Ashman and Hull (32, 33) used the following formula  $QI = K \log_{10}(R - R + 0.07)$  in which  $K$  was 0.385 for males and 0.375 for females.

The duration of mechanical ventricular systole in man was studied by Lombard and Cope (962), using the electro pulse tracing which in fact is an index of ventricular ejection time rather than of total contraction time. They emphasized the large influence of the volume of venous inflow on the duration of ventricular systole and the negligible and inconsistent influence of arterial pressure (systolic pressure, diastolic pressure, or pulse pressure). They recognized that the heart sound method is probably the most accurate for determining this value. The duration of systolic ejection as measured by Lombard and Cope was shortest in the standing position, longer in the sitting, longest in the recumbent—the expected effects of difference in venous filling. Height, weight, age (15 to 65 years), season of the year, and use of tobacco had no apparent influence. Women had longer systoles, particularly for their rates. Because of heart to beat variability, which one would suspect was probably largely respiratory in origin it was found necessary to use the average of at least 15 cycles.

Lombard and Cope (962) found that the relation of the length of systole to cycle length could be described by a straight line formula but suspected that the formula does not apply at rates in excess of about 120 per minute. The formulas they derived were as follows ( $L$  = cycle length,  $S$  = length of systole)

|                         |                       |
|-------------------------|-----------------------|
| Men, standing at rest   | $S = 0.150C + 0.1212$ |
|                         | $S = 0.147C + 0.1478$ |
|                         | $S = 0.10C + 0.2010$  |
| Women, standing at rest | $S = 0.157C + 0.1475$ |
|                         | $S = 0.135C + 0.1683$ |
|                         | $S = 0.06C + 0.2478$  |

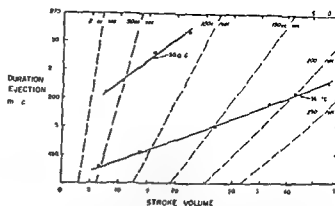


Fig. 79 Relationship between stroke volume and the duration of systolic ejection per beat (as well as the consequent mean rate of ejection—dashed line). Heart rate constant at 115 per min and mean aortic pressure constant at 100 mm Hg in dog heart preparation of Sarnoff and collaborator (1118). Solid dots indicate values obtained at 36.2°C and open circles those at 30.0°C (Courtesy of Braunwald, Sarnoff and Stunby and of Circulation Research (171)).

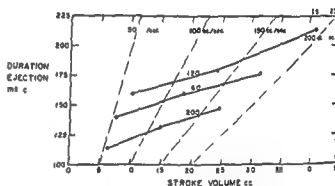


Fig. 80 Influence of heart rate on the relationship between stroke volume and systolic duration. Conditions otherwise the same as in Figure 79 (Courtesy of Braunwald, Sarnoff and Stunby and of Circulation Research (171)).

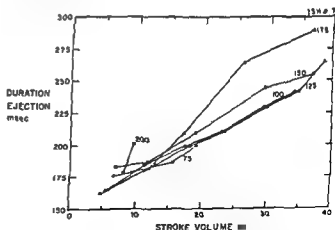


Fig. 81 Influence of aortic pressure on the relationship between stroke volume and duration of ejection. The numbers by each line indicate the pressures at which the values were obtained (Courtesy of Braunwald, Sarnoff and Stunby and of Circulation Research (171)).

Levin Golblin and Burge (92a) used the formula

$$K = \frac{D}{C(C+1)}$$

in which  $D$  and  $C$  were diastolic period and complete cycle length in seconds respectively, as derived from the arterial pulse pressure curve.  $K$  was normally 0.004-0.0049. Long systoles gave values for  $K$  below 0.004. Short systoles gave value above 0.0049. The values for  $C$  and  $D$  were obtained from the arterial pulse pressure curve.

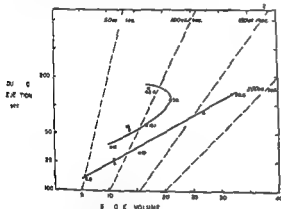


FIG 87 Relation ship between stroke volume and duration of ejection (a) in the presence of a descending failure limb of the ventricular function curve (open circles) and (b) after the administration of 5 mg meyhenthermine sulfate (Wyamine) (solid dots). Numbers contiguous to the points indicate left ventricular end diastolic pressure in cm H<sub>2</sub>O. (Courtesy of Braunwald, Sarnoff and Stein by and of Circulation Research (11).)

TABLE 5

The hemodynamic determinants of systolic duration

|   | Duration of Ejection per Beat | Duration of Ejection per Minute | Stroke Volume |
|---|-------------------------------|---------------------------------|---------------|
| Increased stroke volume                           | ↑                             | ↑                               | ↑             |
| Failure limb of Starling curve                    | ↑                             | ↓                               | ↑             |
| Increased heart rate with constant stroke volume  | ↓                             | ↑                               | ↑             |
| Increased heart rate with constant cardiac output | ↓                             | ↑                               | ↓             |
| Hypothemia  | ↑                             | ↑                               | ↓             |
| Sympathomimetic amine                             | ↓                             | ↓                               | ↑             |

From Braunwald, Sarnoff and Stein by (11)

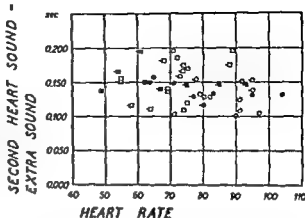


FIG 83 Relation of S-T-S to heart rate

Squares male Circles female Physiologic sounds in black pathologic open (From Frost (48)). There is a slight trend toward a shorter interval at higher heart rates a finding consistent with that shown in the next figure.

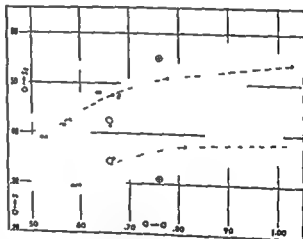


FIG 94 Relation of Q-S<sub>2</sub> and Q-S intervals to cycle length

Data from 70 cases of normal and diseased hearts in persons from the age of 11 to 70 years. The large open circles are measurements in a patient with constrictive pericarditis before surgery and the large closed circles after surgery. The chart indicates the fact that before operation the protodiastolic sound was closer to S<sub>2</sub> than in the average case and that the interval increased after operation. It is of interest that systole was abbreviated after surgery. (From Dunn (35?))

Mechanical systole is measured by the heart sounds is normally very slightly shorter than electrical systole as gauged by the QT interval. Mannheim (1031) found that electrical systole was 21 per cent longer in normal children and 34 per cent longer in children with congenital heart disease.

There is much clinical and experimental evidence that venous filling has an important effect on systolic duration. Systole is shorter when the subject is in the standing position. The duration of systole in each ventricle can vary independently of that in the other ventricle. With inspiration the duration is increased in the right ventricle and decreased in the left—changes which parallel those in venous filling. In atrial septal defect systolic duration is usually consistently longer in the right ventricle, in patent ductus arteriosus the left ventricle tends to have a longer systolic period. There is lengthening of the systolic period in the first normal beat after the compensatory pause of an extrasystole. Systole is longer with the mitral venous infusion of saline and is briefer than is normal for the rate during shock. The lengthening of systole at slow heart rates, in complete heart block for example, is probably related to the increased stroke volume, the enlarged heart and large stroke volume of complete heart block induces increased diastolic filling of the ventricle.

In a stable dog preparation (1348) in which they could vary venous filling Braunwald, Sarnoff and Stumphy (164, 171) found a linear relationship between stroke volume and duration of systole. See Figures 79 to 82 and Table 5. Increased mean aortic pressure had little effect until high levels were attained. At pressures approaching 200 mm Hg a prolongation of systole was observed. This finding is in disagreement with that of Wiggers (1553) who found no abbreviation of systole with acute elevations of aortic pressure.

In heart failure digitalis which reduces diastolic volume of the heart reduces the duration of systole (254) despite the lengthening that might be expected from the bridgehead effect of this drug. In mitral stenosis Wells (1527) claims that

ventricular systole is abnormally short because of reduced stroke volume. (Of course, the delay in the mitral first sound (see p. 286) causes the systole to be even shorter.)

The physiologic third sound occurs normally about 0.14 sec after the beginning of the second heart sound. Frost (498) found in adults a mean value of 0.147 sec ( $SD = 0.025$ ). Minnhimer (1031) found shorter values in children (see Table 2). It occurs during the phase of rapid ventricular filling as indicated by the downstroke beyond the V peak of the venous pulse (the V limb). One study found a seeming temporal relationship between  $S_3$  and the peak of the V wave of the jugular venous pulse and interpreted this as indicating that  $S_3$  is an opening sound of the AV valves. The temporal relationship is probably spurious and the result of time lag in the jugular venous pulse wave (828). Frost concluded that the interval between  $S_2$  and  $S_3$  is not influenced by heart rate or sex (497, 498) but does vary with age (983)—probably as a function of heart size—becoming longer at older ages (Fig. 83). Dunn (382) presents evidence for a change in  $S_2$ - $S_3$  interval with heart rate (Fig. 84). A systematic study of the relation of this interval to the length of the preceding diastolic period in sinus or rhythm or in atrial fibrillation remains to be done.

The atrial heart sound usually occurs about 0.12 to 0.17 sec after the beginning of the P wave. Frost (498) found a mean value of 0.141,  $\sigma = 0.023$  and 0.05 to 0.09 sec after the onset of atrial systole is signalled by the rise of mitral atrial pressure in the cardiac catheter curve. Courmand (310) found in eight adults an average latent period of 0.11 sec between the onset of the P wave and the onset of the right atrial pressure wave.

## CHAPTER 10

# The Generation of Sound in the Cardiovascular System

Although it is occasionally difficult to draw the line between the two, cardiovascular sound can be divided into (1) circumscribed sound or transients (actually complexes of transients in some cases) the heart sound and (2) longer combinations of vibrations usually called murmur (or occasionally especially in the case of peripheral vascular murmurs *bruits* or *souffles*).

*Mechanism* is a flexible term and concept like the word *basic* which is sometimes combined with it. In the following discussion when it is stated that a given sound is due to valve closure or that a given murmur is due to retroversion of a valve cusp it is recognized that it may be more exact to state that sound is associated with a particular event or anatomic set up. It is recognized that when the mechanism of a sound is given the detail of mechanism of the generation of the sound have not been elucidated.

### TRANSIENTS

The first and second heart sound are in the main valve closure noises (339-910). The first sound is associated with closure of the two atrioventricular valves and the second sound with closure of the two arterial valves. Current thought has been along the line that tensing of the valve curtain not collision of the margins of the cusp is the basis for the sound produced. Doek like Rouinet in the early 1830 has done experiments tensing valve segments with the production of noises like heart sounds. Dr Francis Palfrey in teaching physical diagnosis at Harvard Medical School was in the habit of tensing his pocket handkerchief to produce sounds like the heart sounds with a greater length of handkerchief for the first sound than for the second (1176).

Since in two the valve curtains are loaded by

blood experimental tensing of fission pericardium or valve segments in air is not completely analogous. This fact was recognized by Doek (337) who had his experiment arranged so that the tensed strips were submerged in water.

It is probably most accurate to think of sounds such as the first and second sound and the mitral opening snap as hydrostatic pressure transients produced by the abrupt interruption of the momentum of a local flow (Fig. 83) (e.g. before closure of the atrioventricular valves there must be slight local flow accompanying the billowing of the valve curtains toward the atrium). The valve curtains and the chordae which support them are inextensible collagenous structures. When their limit has been reached they interrupt the local flow abruptly with a translation of energy into a pressure transient.

In the case of the closure of the arterial valve resulting in the second heart sound there usually appears to be a local backflow toward the bellies of the valve cusps. The interruption of this backflow again incites a pressure transient which after transmission to the surface of the chest and detection by appropriate means is designated the 'second heart sound'.

In connection with the point of view just explained it should be noted that the pressure transients are located predominantly in one chamber or vessel. The first sound in the ventricles the second sound in the base of the aorta or pulmonary artery the mitral opening snap (see below) in the left atrium etc. This fact accounts for the findings of intracardiac phonocardiography particularly the pronounced attenuation from one chamber to the next and the fact that  $S_1$  is loudest in the right ventricle whereas the second sound is loudest in the pulmonary artery.



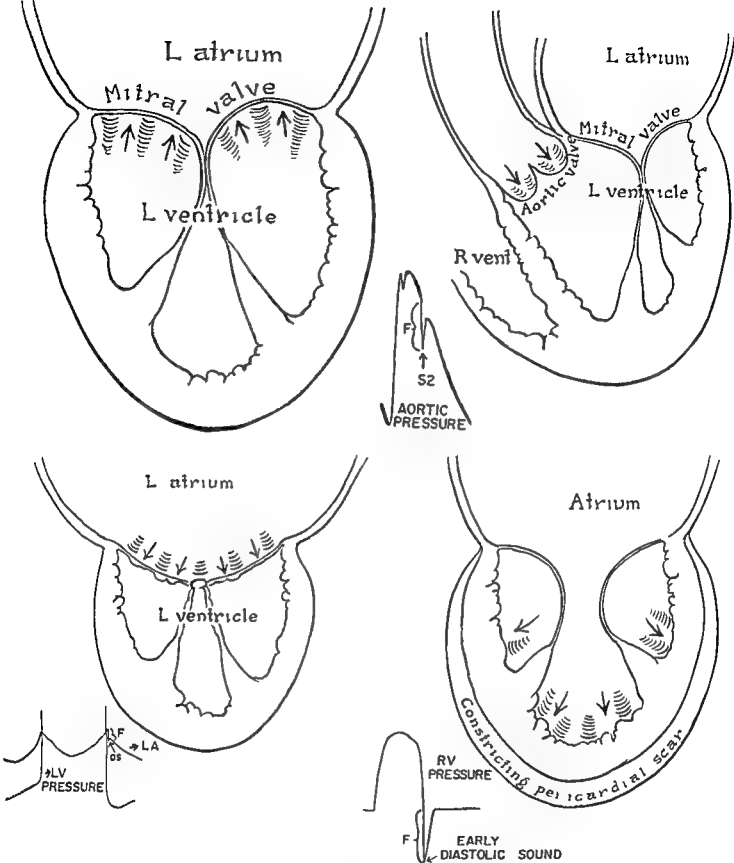


FIG. 85 A schematic representation of the inciting of pressure transients when a valve in closing brings in abrupt halt to local flows. (Left top) Mitral (or tricuspid) closure. The aortic valve has not yet opened. Note that the pressure transients are conceived as being created mainly in the ventricle. The resulting sound is heard mainly at the apex. (Right top) Aortic (or pulmonary) closure. The pressure transients are conceived as being created mainly in the great vessels. The resulting sound is heard mainly at the base of the heart. (Left bottom) Mitral valve opening in mitral stenosis. The pressure transients are conceived as being created mainly in the left atrium. The resulting sound is heard mainly at the left sternal border but also at the aortic area and in the suprasternal notch. (Right bottom) The early diastolic sound in constrictive pericarditis. Pressure transients are generated when ventricular flow is brought to an abrupt halt by the constricted ventricular wall after the ejection of the ventricle has been attained. In pressure curves (inerts) F = time of local flow, arrow = time of sound.

(946) Furthermore it accounts for some of the topography of the valve sounds on the surface of the chest. Is it any wonder for example that the first sound is loudest at the apex and the second sound at the base?

The above point of view is stated by Rühmer (1932) as follows:

Since the chambers of the heart are filled with blood none of these structures can vibrate independently without producing movement of the blood. Similarly vibrations in the blood must be transmitted to the surrounding structures. If the sound can be picked up from the external surface of the body, all structures between the heart and the thoracic wall must be vibrating. It is futile to consider vibration of the heart wall, valves, arterial wall, and blood individually when in fact they constitute an interdependent system and all vibrate at the same time. A more realistic approach to the problem results from considering those conditions which lead to vibrations of cardiovascular system composed of the blood, heart walls and valves.

Rühmer suggests that the heart sounds are the result of abrupt acceleration or deceleration. The valve closure sound (the mitral opening snap and the sound of constrictive pericarditis) are seemingly the product of abrupt deceleration of local flow. Whether abrupt acceleration as with valve opening can produce sound is less convincingly demonstrated but clinical experience would suggest it can. The sound generating properties of opening valve should be studied in models simulating the heart.

Kuwahara (709) also seems to have conceived of the same general mechanism outlined above in analogy to the hydraulic ram although ingenious may not be entirely applicable.

The students of conventional phonocardiography have divided the heart sounds into several components to each of which a separate origin has been assigned. The likelihood of fallacy in assigning epiradi significance to individual quiggles in an oscillogram is a priori enormous. Furthermore if one divides the heart sounds into four components as has been fashionable then one should divide them into eight inasmuch as there are really two multaneously functioning heart each capable of producing the four types of noises (862).

In the case of the first heart sound components have been ascribed to the contraction in the myocardium itself and to opening of the arterial

valves and/or to movement of blood out of the ventricle. At the onset of the first sound there are vibrations which have been thought to be the residual effect of atrial contraction. This complicated schema has little evidence to support it. Although it is long established that the contraction of skeletal muscle (499, 677) and presumably of myocardium is productive of a sound the sound must be of low frequency and intense.

Most recently Lurida (982) has championed the existence of four main components in the first sound and relates these to mitral closure, tricuspid closure, aortic opening and pulmonary opening. I wish he thinks. A valve opening is noiseless because it is occurring at low pressure. On the other hand he thinks that the opening of the arterial valves at high pressure causes a clicking which is expressed as a sound. Intracardiac phonocardiography and animal experiments provide support for the above view. On the other hand Newbold and Rudhe (1257) with electro-kymographic correlations could find support for the existence of mitral and tricuspid components (in that order) in the first sound but none for a contribution by arterial valve opening. They like Lurida concluded that normally A valve opening is noiseless.

There is a low frequency component preceding the sound(s) of closure of the A valves (1139). This was interpreted as representing 'residual atrial vibrations' by Cooley and La Caze (304) by Orús and Bruen Menendez (1166) and by Rappaport and Sprague (1244). Later the observation that these vibrations persist in the presence of atrial fibrillation (908) made this explanation untenable. They occur after the onset of the QRS of the electrocardiogram (908) and although this fact does not exclude atrial origin, they can therefore be related usually to myocardial contraction preceding closure of the A valves. The

vibrations preceding the major component of the first sound in case of mitral tenor probably have a different explanation (see p. 287). In brief the evidence is thought to represent the tricuspid closure sound rendered audible by the paradoxical sequence of mitral and tricuspid closure in mitral tenor. The possibilities exist that some of the recording on the basis of which it was concluded that the initial vibrations persist in atrial fibrillation were made in patients with mitral tenor and that in fact the vibrations in question represented tricuspid closure sound.

low frequency composition of this component is consistent with a muscular origin. Since the right ventricle usually begins to contract first but the mitral valve closes first, the interval between the onset of the "initial vibrations" and the "fast vibrations" of the first sound cannot be used as a measure of Kelly's (779) mechanical lag phase.

The occasional atrial origin of the initial vibrations cannot be completely excluded. Duchosal (377) found that as a patient with atrial gallop improves clinically the interval between the gallop and  $S_1$  becomes less and at times the atrial gallop becomes part of  $S_1$ . Leonard and his colleagues (876) found that in cases of atrial gallop in hypertensive patients application of tourniquets to the extremities causes the atrial sound to move progressively into the first sound and assume the appearance of the initial vibrations. This suggests that the initial vibrations may sometimes be atrial in origin. See also Iran's gallop pre-systolic retardé" (p. 173).

In early systole there is frequently a very short ejection murmur even in normal persons which in the oscillographic phonocardiogram, in particular, may be interpreted as a prolongation of the first heart sound. When aortic stenosis or pulmonary stenosis is present a forward snapping of the stenotic valve membrane or an abrupt cessation in the opening process may produce a snap intimately related to the first sound.

When dilatation of pulmonary artery or aorta occurs, especially if hypertension is also present in the great vessel in question, a click in early systole may occur and may be interpreted as part of the first sound. The *early systolic click* may result from a snapping of the vessel wall with ventricular ejection or, more likely following the observations of Hultgren (725) from a snapping of the inextensible valve ring or annulus fibrous. Hultgren could record no snap directly over the great vessels but could record a snap over the annulus fibrous. The most effective way to produce the sound artificially was to place a band of

inextensible material around the main pulmonary artery or aorta, without producing compression.

It should be pointed out that some believe the early systolic click is an exaggerated form of the normal opening sound of the aortic and/or pulmonary valves. They state that this exaggeration occurs not only with disease of one of the two great arteries but also with either aortic stenosis or pulmonary stenosis. Davidson (330A) goes so far as to suggest a different terminology—pulmonary first sound' (1<sup>st</sup> sound)—for the now current pulmonary early systolic click'.

The students of oscillographic phonocardiography have divided the *second heart sound* also into four components: three in addition to the noise of closure of the arterial valves: initial vibrations of ventricular relaxation, a sound of opening of the AV valves, the after vibration in the great vessels above the valves. The only possibility worthy of serious consideration is that of a normal opening sound arising at the AV valves. In mitral stenosis and tricuspid stenosis such occurs quite as a rule as the so called 'opening snap'. In conditions of high flow across the AV valves the question of an opening snap without actual stenosis has been raised for example Latham (863) has made reference to a tricuspid opening snap in atrial septal defect. Although perhaps a faint AV opening sound will be found on intracardiac phonocardiography in normal it is doubtful that such sound can be detected on the surface of the chest (p. 84). Low frequency vibrations which have been seen at the end of the second sound in oscillograms and ascribed to AV valve opening (983) would seem to occur too close to the second sound to be this judging by the experience with the bona fide opening sound of mitral stenosis.

In connection with the production of the *physiologic third heart sound* and the *protodiastolic gallop* which is generally conceded to represent a pathologic exaggeration of the former, two main suggestions<sup>3</sup> are promulgated: (1) that they repre-

<sup>2</sup> The production of sound by contracting skeletal muscle (399-877) is an old observation (p. 42) and sounds have been recorded from contracting myocardium (1410-1412) but is generally conceded that the noise produced is of low frequency composition.

<sup>3</sup> Proponents for the myocardial origin have been among several Potain, Wolffert and Virgohies (1577) Oras and Braun Menendez (1166) J. R. Smith (1408) and Kuo (828) proponents of the valvular origin have included Hirshfelder (691) Gibson (547) Lewis and Dock (898) Brady and Taubman (149) Dock Grindell and Taubman (361) and others.

sent vibrations set up in the ventricular myocardium by rapid ventricular filling and (2) that they represent a re-closure sound of the AV valve or at least that a reflux with rapid ventricular filling occasions tension of the curtain and/or the chordae of the AV valve with production of the observed sound. The critical experiment has not yet been devised and there are bits of evidence suggesting or contradicting both suggestions. For example in kymographic studies of ventricular border motion in patients with a protodiastolic gallop Brady and Taubman (149) described an anomalous medial motion at the time of the gallop sound. They presented this observation as evidence of the reflux necessary to the second theory. An extracardiac contribution to the sounds was suggested by Dunn (152) who reasoned by analogy to the early diastolic sound of constrictive pericarditis and by Harvey (652) who in describing what he termed ventricular knock and what is almost certainly merely an exaggerated protodiastolic gallop occurring in association with mitral regurgitation observed a striking impact of the heart against the anterior chest wall.

At various times the view has been expressed that the physiologic third sound is a physiologic opening snap of the AV valves. As recently as 1954 (402) a study which appeared to support this notion consisted of correlation of the jugular venous pulse with the heart sounds. Lag in the former (829) probably accounts for the seeming correlation between the peak of the V wave and the third sound.

The idea that the third heart sound arises largely in the ventricular myocardium is attractive. It would not seem inconsistent to think that tightening of the chordae and/or cup of the AV valves also occurs as the ventricle elongates and contributes to the third heart sound. The variation in quality of the third heart sound—from a dull thudding sound of low frequency in the S<sub>1</sub>CC to a sharp even snappy sound with correspondingly different pattern in the S<sub>1</sub>CC—is consistent with a dual mechanism and varying proportions of the two contributing factors. Orris (111) also points out that in locating the origin of the vibrations in the ventricular wall one is not

excluding tendinous and valvular structures from participation in the tensioning process.

One bit of evidence which is usually overlooked in discussions of the genesis of the third heart sound and which is against the purely valvular origin of the sound—at least against re-closure as the mechanism—is provided by Ryd and demonstration (1336) that in atrial fibrillation S<sub>3</sub> is loudest when it occurs in that part of the cardiac cycle in which the normal third heart sound occurs (see Fig. 447 p. 437). This would suggest that the AV valves are flung most widely open at this time. To be sure an alternative explanation agreeable to the valvular school is that there is summation of valve closure sound—the ordinary third heart sound and the first heart sound—to account for the observations noted.

It must be pointed out that re-closure in part or in whole of the AV valve has not been excluded although it also has of course not been established. Reversal of the pressure gradient across the tricuspid valve was found by some workers (876, 956) but not by others (825) (828).

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Regardless of the details of mechanism two factors seem to be involved in the production of the protodiastolic gallop: (1) rapid ventricular filling and (2) alteration in the volume characteristics of the ventricle.

There is some difference of opinion as to whether a protodiastolic gallop can be produced in the right ventricle. In the experience of Wolferth and Margolies (1577) three of 60 gallops were right-sided. On the other hand Frost (409) seems to have thought that all gallops are produced in the left ventricle. The occurrence of gallops with cor pulmonale and with pulmonary stenosis would seem adequate evidence that the right ventricle can produce gallop sounds.

In constrictive pericarditis there occurs in early diastole a sound which is usually more closely situated to the second sound than either a physiologic third sound or a protodiastolic gallop. This sound can be circumstantially evidenced in the form

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In constrictive pericarditis there occurs in early diastole a sound which is usually more closely related to the second sound than either a physiologic third sound or a protodiastolic gallop. This sound can be circumstantial evidence in the form

of correlated recordings (see Fig 42a), be demonstrated to be related to abrupt halt in ventricular filling. It is a "water hammer" sound or "cocktail shaker" sound. Most students now hold that this sound is fundamentally the same as the protodiastolic gallop and that its unusually close position to the second heart sound is merely a function of the unusually high venous pressures—and therefore unusually rapid ventricular filling—which are features of constrictive pericarditis. However, almost never in congestive heart failure on other bases does one encounter a protodiastolic gallop quite as close to the second sound even with venous pressures in a comparable range. Therefore, the limitation on diastolic filling with an abrupt halt in filling must be a factor in the timing of the sound. The protodiastolic gallop of pericardial effusion probably has a similar basis—impact vibrations are produced when the capacity of the ventricle is attained. The protodiastolic sound of constrictive pericarditis illustrates the production of a sound from abrupt deceleration (see Fig 85D).

The atrial or fourth heart sound occurs normally in children and occurs in exaggerated form in adults as a presystolic gallop or in association with heart block. In connection with this sound there are protagonists for a myocardial origin and those for a valvular origin, as in the case of the protodiastolic sound.

Oscillographic studies reveal two separate components to the atrial sound, each probably with its own mechanism. The first element is always audible in the esophagus and probably is related to tensing of the contracting atrial wall. That the sound indeed has this basis may be supported by the facts that an atrial sound may be found in the midst of ventricular systole in cases with nodal rhythm and retrograde atrial excitation (376) and in cases with complete atrioventricular dissociation (346). The second element is recorded mainly from the precordium and probably is a sound generated in the ventricular myocardium or possibly the AV valve mechanism as a secondary result of the contraction of the atrium.

The unusually snappy character of the atrial sound in many cases of presystolic gallop may indicate that tensing of the AV valves is involved. Henderson and Johnson (670) in the now classical

paper in which the role of atrial systole in closure of the AV valve was defended, went much further than more recent evidence would permit and insisted that closure of the AV valves occurs normally in presystole and that only with atrial fibrillation and other situations in which atrial contraction is missing, at least from the usual position in the heart cycle is there what they thought of as a "hinge mechanism" of closure of the AV valve through elevation of ventricular pressure. This idea is still consistent with the view that the first heart sound (which occurs after the onset of ventricular contraction) is due to tensing of the AV valve, since this tensing could occur even in a closed valve. However, the absence of an audible atrial sound except in a small number of young subjects—it can be demonstrated in recording from the esophagus in all persons—seems against a complete closure of the AV valves with atrial systole under normal circumstances. The possibility certainly remains that such occurs with unusually strong atrial contraction, that is, in the conditions in which a presystolic gallop occurs.

Leonard and co-workers (876) believe that the presystolic gallop of arterial hypertension occurs earlier than the normal atrial sound of which it is a pathologic exaggeration. The normal atrial sound may occur so late as to fall after the onset of the QRS. What is interpreted as a muscular component of  $S_1$  may be in fact an atrial sound in some instances (see p 126). By cutting down on venous return to the heart by venous pooling, Leonard *et al* (876) could demonstrate that the presystolic gallop will decrease in intensity and move progressively toward or even into the first sound. Seemingly venous pooling has normalized the situation with respect to both hemodynamics and heart sounds. The audibility of the exaggerated and abnormally early atrial sound in systemic arterial hypertension is enhanced by delay in the first heart sound (p 427).

In discussions of the mechanism of gallop sounds it is likely that a careful distinction should be made between the two types. We do not know that the mechanism of the two is identical. Furthermore, there is some reason to suspect it is different since the clinical setting for the two is in a general way different (see p 175).

*Clicks occurring in systole* are believed to arise

in extracardiac structures for the most part. The early systolic click of dilated aorta and pulmonary artery particularly with hypertension in the vessel in question appears to arise through snapping or tensing of the vessel wall early in ejection. Hultgren (72) presents evidence in support of the view that it is snapping of the mitral valve ring which is responsible for the sound. Leatham and Vogelpoel (86a) suggested an identity to the ejection vibrations recorded by Wiggers and Dera (1934) on the exposed great vessel of the dog. Finally Lunsford and colleagues (982) insist that the early systolic click is an exaggerated form of the sound which they think normally occur with opening of the arterial valves and constitutes the latter part of S<sub>1</sub>. What they apparently conceive of as vibration initiated when the ejected blood first meets the static blood in the base of the aorta.

Mid and late systolic clicks are commonly produced by the tensing of pleuropericardial adhesions. Supporting this statement the evidence such as it is is follows: (1) These clicks are often observed to appear following an attack of acute pericarditis. (2) They may occur in patients with extensive pleural and pulmonary disease by tuberculosis (Fig 141) sarcoid (761) etc. (3) Pleuropericardial adhesions have been observed at necropsy in patients who manifested clicks in life. (4) Lunsford (981) observed on fluoroscopy a tugging at one leaf of the diaphragm synchronous with the systolic click. It is difficult to understand why the click or clicks should be late in systole when this is the mechanism. Sometimes the systolic click initiates a systolic murmur which is usually noisy but sometimes musical and which is believed to result from the rubbing together of roughened pericardial surfaces. Although the early systolic click is most often produced in the aorta or pulmonary artery, pleuropericardial adhesions at times produce an early systolic click. Furthermore a click with this mechanism may occur in early diastole (761). Usually however there are in such case other click(s) in late systole.

Although the above arguments for pleuropericardial adhesion as the basis for systolic clicks has failed to convince some (604A) (1979) most would agree that they are of extracardiac

source. They bear no constant relationship to one part of the cardiac cycle. Their position varies from early systole to early diastole and is influenced by respiratory phase and position of the subject.

Sometimes mid systolic clicks appear to be due to movement of costochondral or chondro-sternal joints by an enlarged heart, or by a normal heart which is in abnormally intimate contact with the bony cage of the thorax because of pectus excavatum or other deformity of the chest or of displacement by a celiac pregnancy or mitrosternal mass. Loose jointedness in the Marfan syndrome may contribute to the production of these sounds.

It is debatable whether there is any category of transient located between the first and second sound other than the clicks produced through the mechanisms mentioned above. Specifically is there anything which can suitably be called a systolic gallop? Occasionally we have observed in cases of aortic regurgitation a mid systolic transient of lower frequency content slightly greater duration and in general more thudding quality than the usual systolic clicks. The mechanism is obscure. It may be produced in the aorta when full capacity is reached. The large stroke volume of aortic regurgitation may account for its occurrence in association with this valve lesion.

There remains to discuss one more category of transient namely the opening snap of the Aortic valves. Lunsford has thought that small vibrations recorded at the surface of the chest can at times be related to opening of these valves in normal subjects and that in intracardiac phonocardiogram such vibrations can be frequently identified. Opening of the mitral or tricuspid valve may be associated with a striking snap when stenosis of the valve is present. The adhesion of the commissures in the cusp results in a fibrotic diaphragm which balloons or better snaps toward the ventricle or toward the atrium when the polarity of the atrioventricular pressure differential is reversed (Fig 86). Abrupt ballooning of the membrane toward the atrium (as well as tensing of the shortened, fibrotic chordae tendineae) contributes to the snappy first heart sound of mitral stenosis. Ballooning in the opposite direction in early diastole produces the mitral opening snap.



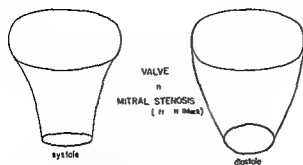


FIG 86 Mechanism of opening snap

The abrupt billowing of the mitral curtain toward the atrium early in ventricular systole and toward the ventricle in early ventricular diastole is a possible explanation for the snapping first sound and the mitral opening snap respectively in mitral stenosis (Adapted from Holldack (701) )

Theoretically the opening snap might be expected to be more intense in the left atrium than in the left ventricle. The pressure wave created when the inextensible aortic leaflet of the mitral valve brings local flow abruptly to a halt is located in the left atrium. The characteristic location of maximum audibility of the mitral opening snap on the surface of the chest is consistent with the theoretical consideration. If it were not for the fact that the pressure transient produced by closure of the mitral valve is located predominantly in the left ventricle and the pressure transient produced by opening of the mitral valve located predominantly in the left atrium, it would be difficult to explain the differences in location of maximum audibility of  $S_1$  and the opening snap—sounds generated at the same structure. An analogy has been drawn to a child's toy "cricket" in which a metal tongue which is stiff, yet flexible produces a snap when pressed with the thumb. The analogy is not appropriate since the anterior (aortic) leaflet of the mitral valve does not have stiffness comparable to that of the tongue of the "cricket." Bending of the metal is rather directly responsible for the sound in the cricket whereas in the opening snap the primary event is the pressure transient(s) created in the fluid when there is abrupt deceleration of local flow by the inextensible valve curtain (See Fig 85C)

Snappy sounds occurring shortly after the second heart sound and possibly representing opening snaps of normal valves occur in instances of very large diastolic flow across the valve such as

the tricuspid valve with atrial septal defect or the mitral valve with ventricular septal defect and patent ductus arteriosus. This is, however, as yet an inconsistent and far from established phenomenon. It is somewhat clearer that an opening snap can be produced in an AV valve which is not stenotic but which is scarred as a result of rheumatism or of fetal fibroelastosis.

## MURMURS

Most murmurs are noises, the genesis of these will be discussed first. Other murmurs are musical, the special features of the genesis of these will be discussed later.

The ideas that will be developed in the following discussion are as follows:

1 Turbulence and murmur cannot be equated to each other in any direct manner. The Reynolds number provides information only of qualitative and descriptive nature and is largely of conceptual usefulness. The Reynolds formula is useful largely as a catalogue of factors involved in the genesis of murmurs.

2 Most murmurs arise through a complex interplay of disturbed flow and the wall and other boundary structures.

3 Cavitation should be considered as a possible mechanism in murmur production.

After a survey of basic physical principles pertinent to murmur production, the classic concepts of the generation of murmurs will be reviewed.

Thrills and murmurs are basically the same phenomenon sensed in a different manner. Therefore what is said about the genesis of murmurs applies to thrills also. One frequently hears murmurs without being able to feel a thrill, but whenever there is a true thrill felt there is also a murmur, usually a loud murmur. The frequency response curves of the fingers for palpation of thrills and of the ear for the hearing of murmurs are different. Palpation is most efficient in the low frequency range, audition in the higher frequency range.

**TURBULENCE** The reader is referred to pages 47 and 48 for description of the experiments on the basis of which Reynolds defined the factors responsible for transition from laminar (streamline) to turbulent flow. The Reynolds formula is as follows:

Reynold number ( $R_N$ ) =  

$$\frac{\text{diameter of conduit} \times \text{velocity of flow}}{\text{kinematic viscosity of fluid}}$$

(kinematic viscosity is absolute viscosity divided by density) The Reynolds number is dimensionless. Furthermore, it represents a law of similitude. Providing that the conditions of the basic experimental model on which it is based obtain, the transition from laminar to turbulent flow will, for example, occur at a Reynolds number of about 2000 for a wide range of flow velocities, varieties of fluid as far as viscosity and density are concerned, and sizes of tubes.

The difficulty encountered in attempting quantitative application of the Reynolds formula to the circulation is that the conditions of the basic model do not obtain. The interior of the heart and base of the great vessel in the vicinity of the valve—sites of greatest interest from the standpoint of murmur production—are obviously quite different from the model. The aorta too is far from a long straight tube of uniform diameter (Fig. 87).

However, there is a sizeable body of evidence that the Reynolds formula is of some predictive value as to the influence that changes in the Reynold parameter will have on the intensity

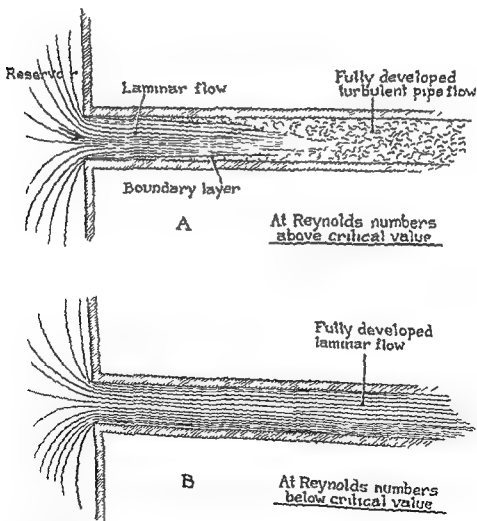


FIG. 8. The drawing illustrates the entrance length before the flow is turbulent or laminar is established. In the case of laminar flow, the drawing attempts to represent the parabolic velocity profile with most rapid flow at the center of the pipe.

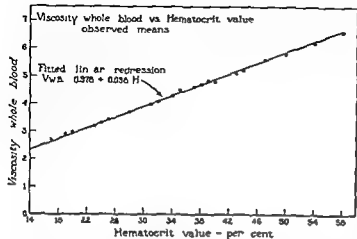


FIG 88 A Relation of blood viscosity to hematocrit (From Nygaard Wilder and Berkson (1149) )

of existing murmurs or the occurrence of murmurs not normally present

1 Reduction in kinematic viscosity in anemia (Fig 88A) is attended by the development of murmurs

2 Increase in kinematic viscosity in polycythemia or hyperproteinemia (Fig 88B) suppresses murmurs which would be present with normocythemia and normal levels of serum proteins

3 Localized dilatations in vessels, such as in aneurysms of the aorta, are the site of murmur production. Dilatation of the ventricle beyond an AV valve ring of normal dimensions is a comparable situation

4 Increased velocity of flow—and increased volume of flow through a conduit of normal caliber which amounts to the same as increased velocity of flow—is accompanied by the production of murmur in thyrotoxicosis, with exercise or injection of epinephrine. This factor is probably also operating in the case of anemia. The increased volume of flow across a normal AV orifice—the tricuspid orifice in atrial septal defect, the mitral orifice in ventricular septal defect and patent ductus arteriosus—is likely to be accompanied by murmur (Dilatation of the ventricle probably contributes in these instances.) Increased stroke volume in aortic regurgitation often produces a systolic murmur in the aortic area. Increased flow across the mitral valve in mitral regurgitation may produce a diastolic murmur or exaggerate the murmur of any mild mitral stenosis which may be present.

The venous hum (see p 226) illustrates very

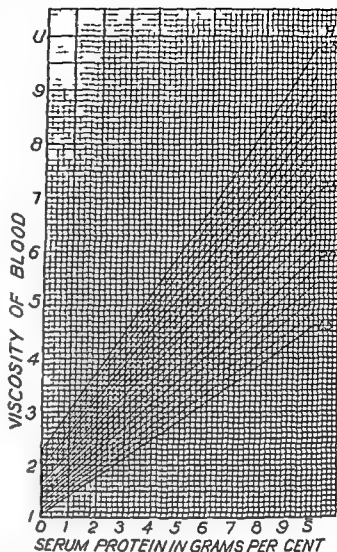


FIG 88 B The relation of blood viscosity to the concentration of plasma proteins (From Limport (816) )

clearly the application of Reynolds critical velocity concept to the generation of murmur. Flow in the veins is basically streamline (666, 667) may become turbulent with appropriate change in any of the factors in the Reynolds formula and murmur develops.

That turbulence is not the whole story in murmur production and that turbulence and murmur cannot be equated to each other directly is suggested by the fact that in tubes with rigid walls Reynolds numbers far in excess of the critical or transitional value are attended by little or no murmur (879). However a murmur does occur when the same flow occurs in a tube with walls of a flexible material such as rubber or plastic. For a murmur of appreciable intensity to be produced the boundary structures must be flexible.

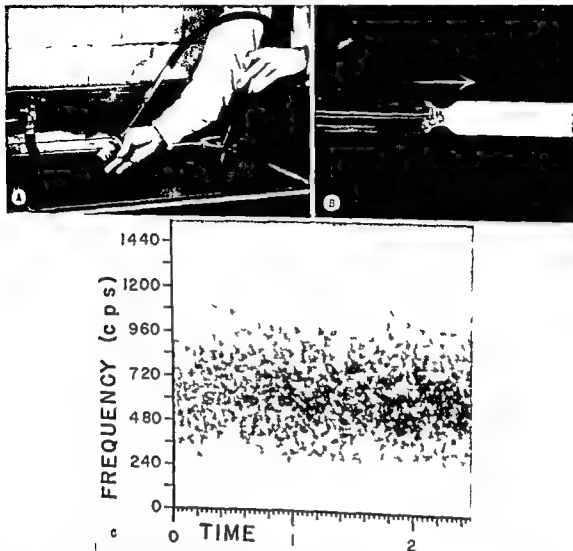


FIG. 5. (A) When water is caused to flow through a tube with a constriction (1) if the velocity of flow is sufficiently high turbulence will form at the neck (B) and produce a humming noise of which the sound spectrogram is shown in (C). The level of frequency displayed by the cavitation noise generated in this rigid walled tube is in the upper range of that seen in some murmurs.

enough to vibrate in response to disturbed flow in the lumen of the vessel or heart.

McDonald (1960) suggests that vortices or eddies should be distinguished from turbulence and that the former may be representative of the

character of the flow disturbance at diseased valve orifices associated with murmurs.

**CAVITATION.** The reader is referred to pages 47 and 48 for a discussion of the historical background of Reynolds' cavitation. Reynolds appears

In 1966 Laurin and Petit (1966) came to a similar conclusion. They indicated that Savart's fluid vein (p. 45) was inadequate to account for murmurs and that eddy oscillations of the fluid veins on boundary structure are responsible.

The experience and experiments relating to the form of cavitation responsible for the findings (Cavitation disease decompression sickness) are pertinent in connection with cardiovascular sound (S. 1963) but cannot answer the direct question of whether cavitation indeed plays a role.

to have been more impressed with this is a mechanism for sound generation in flowing liquid than with turbulence. The structural similarity of the Reynolds tube (Fig. 89) to valve stenoses and concretion of the aorta raises the possibility that the drop in pressure at the constriction in accordance with the principle of Bernoulli may result in bubble formation which in turn is accompanied by murmur production.

The following remarks or thoughts might indicate that cavitation is an unlikely basis of murmurs.

1 In the cardiovascular system there might never be sufficient velocity of flow at constriction to produce the drop in pressure necessary for bubbling.

2 If the necessary drop in pressure does occur, might it not result in collapse at the neck of the constriction before bubbles would develop? A musical type of murmur by a "flitter" mechanism might be anticipated more than cavitation. (However, "flitter" and "cavitation" are not mutually exclusive phenomena.)

3 Cavitation which occurs in association with improperly engineered ship propeller blades is a violently corrosive and destructive phenomenon. Might not intolerable damage be produced in the walls of the cardiovascular system and the valves? Cavitation damage is avoided by the use of a rubber coating of propellers. The softness of the walls of the cardiovascular system may have a similar protective effect.

4 The large and abrupt change in pressure at the neck of the constriction and the humming effect of the bubbles might be expected to produce disruption of the erythrocytes.

The bubbles which form in the process of cavitation may consist either of dissolved gases or of the vapor phase of the liquid in question—in the case of blood, water. One might think that favoring the possibility of cavitation is the fact that the blood has relatively high partial pressure of gases. In fact cavitation in the form of water vapor appears to occur with greater facility than cavitation from dissolved gases.

Even if Reynolds cavitation does not occur in the circulation, and such indeed seems unlikely, it is possible that cavitation of some special variety does. For example the pressure drop in the center of an eddy, the importance of which is

emphasized by McDonald (1960), might be adequate to initiate bubble formation.

Certain classical mechanisms for murmur production can now be reviewed (Fig. 90).

1 The abrupt widening of the conduit or the resumption of normal caliber beyond a constriction is productive of a murmur. The mechanism here is undoubtedly complex. Turbulence and/or eddy formation in the fluid and, possibly, cavitation are important, the disturbance of flow which these represent causes the wall to vibrate. There may be direct driving of the wall just distal to the constriction through the opposing forces of the wall elasticity and the centripetal force described by the principle of Bernoulli.

2 Free flow at high rates can be accompanied by the production of murmurs. The interplay of turbulence with the vibrating wall is probably the basic mechanism.

3 Jet impact—direct driving of a wall opposite an orifice at which the jet is produced—is probably an important mechanism in patent ductus arteriosus, peripheral arteriovenous fistula, coarctation, aortic stenosis and possibly mitral regurgitation. In some of these situations jet lesions—a localized patch of atherosclerosis—develop at the site of impact of the jet (109).

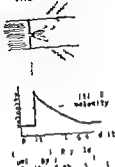
Jet lesions\* occur in many parts of the cardiovascular system when because of acquired or congenital abnormality of architecture there is a small orifice with a high pressure differential on its two sides. Jet lesions are useful anatomic labels for sites of murmur production. In addition to patches of atherosclerosis at the point of traumatization by direct impact, there may be cusp-like pocket-like lesions, sometimes referred to in the older literature as Zahn's or Schmucke's pockets. These appear to represent a scuffing up of the endocardium by the regurgitant stream impinging somewhat obliquely on the surface in question. In bacterial endocarditis organisms may lodge on jet lesions.

In aortic regurgitation, the jet may strike the interventricular septum (most frequently) the aortic leaflet of the mitral valve (with production of an Austin Flint murmur) or the anatomical apex of the left ventricle (with production of

\* I am indebted to Dr. J. I. Edwards, Rochester, Minn. for many details of this section.

## THE MECHANISM OF MURMURS

### THE MODEL I



### EXAMPLE



STENOSIS OF ANY VALVE  
DILATATION OF THE AORTA

### THE MODEL II

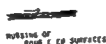


FREE FLOW AT HIGH VELOCITY  
AND/OR LOW VISCOSITY  
REYNOLDS NUMBER IS INCREASED

### EXAMPLES

ANEMIA  
HYPERHIDROSIS  
FEVER  
CONGESTION SUPPRESSION  
OF MURMUR IN  
POLYCYTHEMIA

### THE MODEL III



MURMUR OF  
ROUGH ENDO SURFACES

### EXAMPLE

PERICARDIAL FRICTION  
RUB

### THE MODEL IV



### EXAMPLES

A CURVATURE OF THE AORTA  
DILATED PULMONARY ARTERY  
REGURGITATION AT A V VALVE

### THE MODEL V



JET IMPACT

### EXAMPLE



MITRAL REGURGITATION

### THE MODEL VI



### EXAMPLES

PROMINENT ASTHMA  
INTERMITTENT HEMORRHOIDS  
SQUASHING CARPOTRACHEAL MURMUR  
RETROPERICARDIAL AORTIC CURVE  
AORTIC SITE OBSTRUCTION

### THE MODEL VII



### EXAMPLE

MODERATOR BAND  
(ABERRANT TENDON)  
OF VENTRICLE

PERIODIC SHEDDING OF VORTICES ALTERNATELY  
AND SYMMETRICALLY FROM OPPOSITE SIDES OF  
OBSTACLE WITH REGULAR TRANSVERSE VIBRATIONS  
OF THE CYLINDRICAL OBSTACLE

Fig 90

predominant axillary location of the murmur—the Cole (Cecil) murmur. With aortic regurgitation without stenosis, a jet lesion may develop in the ascending aorta as a result of the re-orientation of the left ventricular outflow tract to the aorta (because of left ventricular enlargement) and as a

result of the large stroke volume. The occurrence of a loud systolic murmur accompanied by systolic thrill in cases of syphilitic aortic regurgitation may be related to this jet lesion.

In mitral regurgitation the most common site of a jet lesion is on the posterior wall just above the

posterior cusp. This fact correlates well with the well known alluvial 'radiation' of the murmur of mitral regurgitation. It is likely that the so called MacCullum's patch (1004) is in many cases a jet lesion rather than a lesion resulting directly from rheumatic fever (174, 408). Edwards and Burchell (412) describe one case in which because of the particular location of the valve lesion in bacterial endocarditis, the jet impinged on the interatrial septum. In this case a systolic murmur and thrill was incorrectly interpreted as indicating aortic stenosis. I have seen cases which are probably identical. The murmur and thrill were maximal in the third right inter-space.

In pure pulmonary stenosis there is likely to be a jet lesion in the region of the bifurcation. In tetralogy of Fallot with infundibular stenosis the jet lesions may be in the infundibulum chamber and on the ventricular side of the pulmonary valve.

In tricuspid regurgitation jet lesions are rare, probably because of a relatively low pressure in the right ventricle but have been described in primary pulmonary hypertension (408).

It is the small ventricular septal defect, with normal right ventricular pressure and high pressure differential across the defect, that is likely to show jet lesions. In brief it is the classical *maladie de Roger* which has such lesions. Since the left ventricle is posteriorly located whereas the right ventricle is the anterior ventricle the jet is directed in the postero-inferior direction. The locale of the murmur and thrill in Roger's disease corresponds to the position of the jet lesion in the right ventricle.

In atrial septal defect pressures are usually not sufficient to produce jet lesions.

In patent ductus arteriosus jet lesions occur in the left pulmonary artery. With the artificial ductus created by the Blalock-Tussig operation the jet lesion is usually located farther peripherally in the pulmonary artery because of the orientation of the subclavian artery in relation to the pulmonary artery.

Edwards has pictured (414) jet lesions of the intima of the aorta distal to a coarctation.

4 Pericardial friction rubs are murmurs too. The mechanism is one of 'stick and slip'. In the usual variety of pericardial friction rub, 'stick and slip' at innumerable points in time and in

location over the pericardium are responsible for the sound. Sometimes by ear and even more clearly by SPCG pericardial friction rubs are seen to be made up of a series of closely spaced clicks. This suggests that in these instances there is a finite number of 'stick and slip' events within, at least, a certain area surrounding the point of auscultation.

*Musical murmurs* differ from noisy murmurs in two striking respects which are probably highly significant with reference to genesis. (1) Considered as a class musical murmurs are more intense than noisy murmurs. Most murmurs audible at a distance from the body are largely musical in character. A colleague of mine heard a

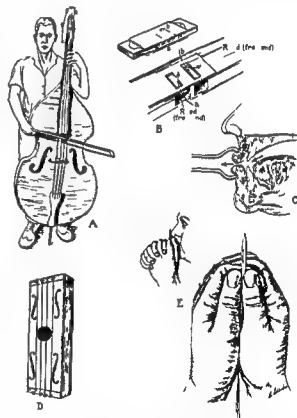


FIG 91 1 Counterparts in musical instruments of generators of musical murmurs.

1 Bass viol (violin family in general) analogous to musical pleuropericardial murmurs. B Harmonica not strictly analogous to retroverted aortic cusp since the reed faces into the wind—not downstream. Reed *a* is activated on blowing and alternate reed *b* on sucking. C Trumpeter's lip analogous to calcific aortic stenosis. D Aeolian harp analogous to anomalous chordae tendinae. E Reed of grass. More like a downstream reed but differs in that the fluid stream passes on both sides of the vibrating member.

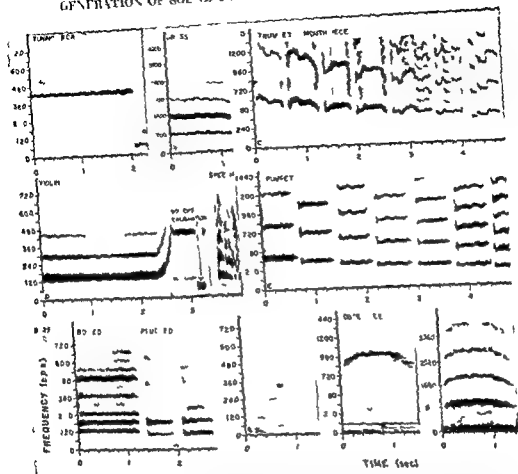


FIG. 91B Spectrograms of musical sounds.

A Tone of tuning fork, 440 cps and on right 60 cps calibration. B Musical sound produced by blowing on a reed of grass. Note that the second harmonic is louder than the first which presumably is the fundamental. This was at the situation in the case of the musical tone produced by a silk thread across a water conducting tube (1887) (See Fig. 91A). C Trumpet mouthpiece. The tones are not steady and impurities in the form of additional partials are present in certain notes. D Violin with portamento (glide) from about 150 cps up to about 440 cps. A brief segment of peech sound; also present and a 60 cps calibration. E Full trumpet. Here the tones are steady and pure. F Musical sounds of a brass instrument. G Recording of musical sounds produced by blowing on a reed. These are only a few of the same sound on three different frequency scale. Below 20 cps there is nothing except noise. The fundamental located at 960 cycles appears in the analysis is at 1440 cps. A total of three overtones are demonstrated in the third analysis.

sea gull' murmur in church. The sound of the patient may complain of the noise in bed (182). (2) For most of the time in association with each variety of musical murmur some elastic boundary structure—stenotic diaphragm valve flap pericardium wall at the tendon cord like structure—which is set into vibration in a specifically periodic and therefore musical manner. Musical tones can be produced by the flow of fluid in a perfectly rigid system. For example the heaving of vortices off the sides of a rigid cylindrical obstruction the so-called Hürmann streets

may produce a musical tone and obviously the flow of air in an organ pipe is productive of a musical tone without the presence of an elastic flap which is free to vibrate. However no such rigid structures occur in appropriately intimate relation to pertinent portions of the cardiovascular system.

In the generation of musical murmurs it is useful to compare the *modus operandi* of the generator to that of various musical instruments (Fig. 91A and B). The physics of musical instruments has been extensively studied and much



of the information so gathered is applicable to musical murmurs.

According to the type of musical instrument most analogous, four general groups of musical instruments are clearly identified, plus a fifth less easily analogized group.

**TRUMPET GROUP** It is possible to play a respectable scale with only the mouthpiece of the trumpet. The function of the rest of the instrument is to introduce purity and control. The anatomical situation in the case of calcific aortic stenosis is analogous. The stenotic valvular diaphragm is analogous to the trumpeter's lip. The pitch of the note produced is determined by the stiffness of the lips, the degree of their separation and the velocity of the fluid stream. Whistling is another analogous situation. Vibration of the lips in whistling can be demonstrated by holding the lips, which stops the whistle.

**AEOLIAN HARP GROUP** Musical murmurs generated at aberrant chordae tendineae, or aberrant tendons ("moderator bands") of the ventricle are analogous to the musical tones produced by the Aeolian harps which in the past were placed in the windows of villas or castles so that the breeze, in blowing through the wires would produce a bit of music. In those cases in which aberrant chordae traverse an interventricular septal defect, the analogy is striking indeed. In association with Chiari's network of the right atrium a musical murmur which is continuous may be produced (see p. 410). Yet another example may be the musical murmur which develops at times over the ascending aorta in cases of dissecting aneurysm and which may be produced by flow past the fibrous cords which traverse the false channel. The murmur in this group are what Bondi (129) referred to as *Fadenge rauschen*.

**REED GROUP** In the case of retroverted aortic cusp, the anomalous member seems to function like the reed of a musical instrument. However, whereas the retroverted cusp opens downstream since it is activated during ventricular diastole by the stream of blood regurgitating into the ventricle, all modern reed instruments have the reed facing into the fluid (air) stream. This is true even in the case of the mouth organ, or harmonica. A blade of grass blown as it is held in

the hands, is a somewhat better analogy, although obviously different because the fluid stream passes on both sides of the vibrating element. In 1886 Sansom (1344), who compared the murmur of calcific aortic stenosis to the whistle produced with the lips, suggested the Jew's harp as a proper analogy for the retroverted cusp. Suggested also by Banks (49) in 1857, it is not entirely satisfactory, either, because of the difference in compliance of the reed and in the mode of production of vibration. Although the analogy to reed instruments does not fit precisely, it seems adequate for characterizing this group of musical murmurs.

**VIOLIN CROWD** Musical extracardiac, i.e., pericardial or pleuroperecardial murmurs are members of this group (1167). The bowed string instruments function by the general principle of "stick and slip" (1238) (see p. 136). The violinist puts rosin on his bow to insure that as he draws it across the strings it sticks with slight displacement of the strings, then, when a certain limit of displacement is attained "slip" occurs and the string springs back toward and past its position of rest. Thus the string is set into, and maintained in vibration. Commonly occurring undesirable examples of "stick and slip" include the creaking of hinges, shattering of window sashes, squealing of tires as a car rounds a corner.

Musical pericardial friction murmurs, either acute and transient or more persistent in nature occur much less frequently than in the noisy variety. However, when such occur one is forced to conclude that the roughening of the pericardium is arranged in a more orderly manner than usual. Possibly the generator area is put into vibration as a whole, in part because it is of limited size rather than there being a great many individual small sound generators operating each at a different frequency as may be in effect the case with a generalized pericarditis.

**"FLUTTER" GROUP** In this category, which can not be analogized to any of the standard musical instruments, the mechanism appears to be flutier a phenomenon which Rodbird (1289, 1291, 1299) has in recent years analyzed in relation to the cardiovascular system (Fig. 92). It is the type of sound produced by blowing through a Sudd valve (Fig. 93) the Bronx cheer has a similar

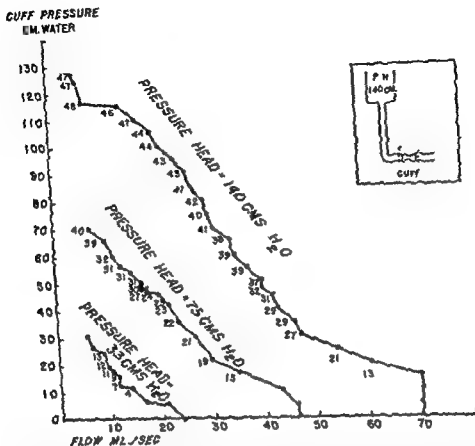


FIG. 92 The Bitter phenomenon

Flow through a soft walled tube is stopped by pressure heads of 140 cm and 35 cm of water respectively. The horizontal axis gives the delivery in ml per sec. The vertical axis represents the degree of constriction of the tube induced by the air pressure in the glass chamber surrounding the soft walled tube. The numbers adjacent to the three lines represent the repetition rate of closing and opening of the tube as determined by stethoscope.

The upper trace shows the change in delivery as the constriction (cuff pressure) is modified in 2 cm steps with a driving head of 140 cm of water. No effect on flow (0 ml per second) is seen at cuff pressures from zero to 20 cm of water. As the cuff pressure increases to 25 cm of water the flow fell to 60 ml per sec and the wall was observed by stethoscope to be closing repeatedly 13 times each second. Increases in cuff pressure further reduced flow and increased the rates of closure in a fairly constant fashion. When cuff pressure was approximately equal to pressure head flow fell below 5 ml per sec and closing and opening of the tube was no longer apparent. Similar data for pressure heads of 0 and 35 cm of water are also given. The data show that the repetition rate becomes faster with either increasing pressure head or increasing degree of narrowing. (From Rodd and (1971))

**mechanism** The bronchial tree in a thin-walled tube is a better example of the bifurcated tube which describes the phenomenon. When an elastic-walled tube such as a bronchus is narrowed to a critical degree the rippling movement is fixed far in the rear of the bronchus) through the narrowed area can be explained by the principle of Bernoulli: such a drop in pressure that the wall at the site of the constriction is pulled in and the lumen further narrowed. But two centrifugal forces tend to enlarge the

lumen: the elasticity of the wall and lateral intraluminal pressure. The result of the two opposing forces—one centripetal and one centrifugal—is that the wall is caused to vibrate in a regular periodic and musical manner. In the arterial and venous systems a situation comparable to that in bronchial tubes probably occurs occasionally so that a venous murmur results.

Certain interrelationships of pressure gradient, flow, dimension of orifice and murmurs



FIG 93 A The Sudd valve used in spirometers and other respiratory equipment

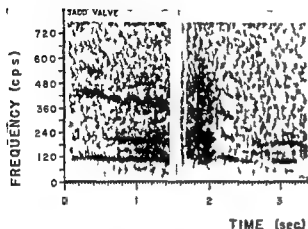


FIG 93 B Partially musical partially noisy sound produced by blowing through Sudd valve

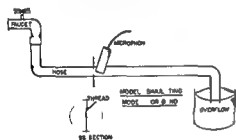


FIG 94 A Model simulating the murmur producing properties of aberrant tendons of the ventricle

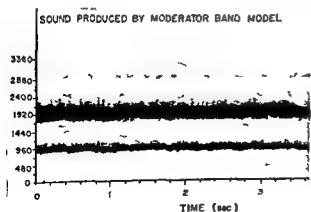


FIG 94 B Murmur recorded from the model shown in the last figure

In schematizing circulatory models of stenosis, Burger, van Brummelen and Dunningburg (197) investigated pulsatile flow and concluded that the application to the heart of a formula for constant flow, as in the case of the Gorlin formula, might not be entirely accurate. With pulsatile flow "a kind of hysteresis phenomenon" not found with steady flow occurred (Fig 94). There was more flow for pressure "on the way up," less flow for pressure "on the way down." Qualitatively, the authors suggested that this could be explained by the fact that the turbulence of the liquid would increase with increasing flow. As time is required for turbulence to set in, there will be a lag as flow increases causing the flow to be less turbulent than would be the case with steady flow at the same pressure differential. The lag in the establishment of turbulence is important and may explain why a murmur does not occur more often in pulsatile flow even though the critical Reynolds number may be exceeded momentarily.

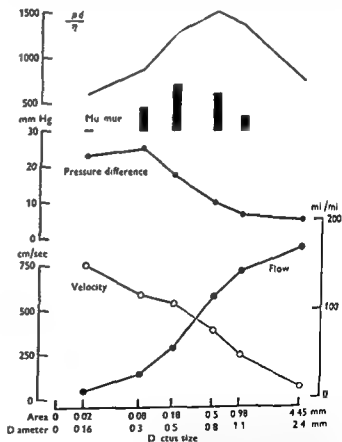


FIG 95 The relationship between the murmur and various hemodynamic parameters in the patent ductus arteriosus of the newborn lamb (From Dawes *et al* (1938))

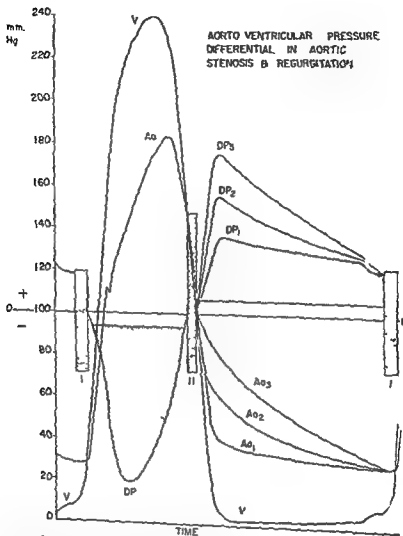


FIG. 3. Pressure gradient and murmurs in aortic valve disease.

The curves displayed here are intended to illustrate certain basic relationships in the genesis of the murmur of aortic stenosis and the murmur of aortic regurgitation. The three main curves are those of ventricular pressure ( $V$ ), aortic pressure ( $Ao$ ) of which three alternative curves in diastole are presented, and the differential pressure ( $DP$ ) of which the three curves corresponding to the three alternative aortic diastolic pressure curves are presented. A basic premise is that the pressure differential across an orifice is intimately related to the intensity frequency characteristics of the murmur produced. From what is known about the shape of the ventricular and aortic pulse pressure curves in aortic stenosis it is evident that the curve of pressure differential in aortic stenosis will have the shape shown in the diagram. The murmur of aortic stenosis has a curve similar as to intensity and peak frequency (the so-called diamond systolic murmur of conventional oscillographic phonocardiography) and the Christmas tree murmur of aortic regurgitation. The configuration of the diastolic portion of the differential pressure curve helps to elucidate several features of aortic diastolic murmurs as they are encountered in practice. Since there is likely to be a certain threshold of differential pressure which must be exceeded before murmur is created, the occlusion in fact rare demonstration of a gap between  $S_2$  and the beginning of the diastolic murmur (SGD) is afforded explanation. Similarly at the end of diastole the diastolic murmur sometimes stops abruptly with the T wave of the electrocardiogram or with the atrial contraction. This phenomenon is probably caused by fall of differential pressure below the murmur threshold as a result of the increment of intraventricular pressure produced by atrial contraction. Within limits the larger the anatomic leak at the aortic valve, the lower the differential pressure and the smaller the diastolic murmur (on first thought a paradoxical phenomenon).

Left heart catheterization is providing actually recorded curves of this type for aortic stenosis and for mitral

—at the peak of ventricular ejection, for example. The faster the rate of cycling of pulsatile flow, the greater was the hysteresis phenomenon.

Other findings of the study by Burger and colleagues (197) included the demonstration that, using stenoses with sharp edges, flow was larger with the same pressure difference if the constriction was longer within limits, e.g., 0.2, 1.0 and 2.0 cm. Flow was maximal in the case of a constriction with sloping profile.

In the study of the parameters of flow in relation to murmur, Dawes, Motte and Widdicombe (338, 339) have made use of the fact that the ductus arteriosus does not close for some days in the newborn lamb. The area of the ductus the

pressure difference between the aorta and pulmonary artery, the intensity of murmur and Reynolds number were calculated (Fig. 95). As might be expected, flow increased steadily with increase in size of the ductus, and pressure difference and velocity of flow fell. Although both the intensity of the murmur and the Reynolds number showed a peak in the mid range of ductus size the curves did not coincide. The peak for the murmur curve lies on the side of smaller ductus size—and, of course, of greater velocity. It is likely that a factor in addition to, or conceivably instead of turbulence is operating in production of the murmur. The other factor may be the effect of a jet from the ductus impinging on the opposite

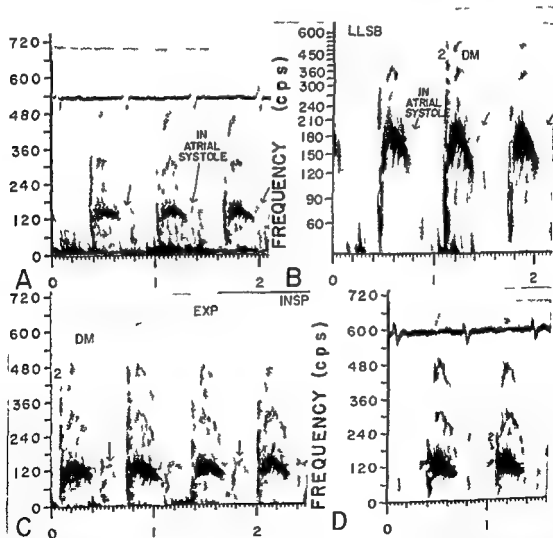


FIG. 97. Relation of shape of aortic murmurs to that of aortic ventricular differential pressure curve.

The curves are all recordings made at different times from the lower left sternal border in a patient with a retroverted aortic cup and aortic regurgitation on the basis of xiphoid. The harmonics of the musical diastolic murmur show the crescendo-decrescendo pattern displayed also in the curve of aortic ventricular pressure differential (Fig. 96). Note that the musical murmur comes to a halt with atrial systole (e.g., D) or is greatly changed (e.g., C). There is a slight harmonic in early systole in C and in late systole in D. B is a logarithmic display.

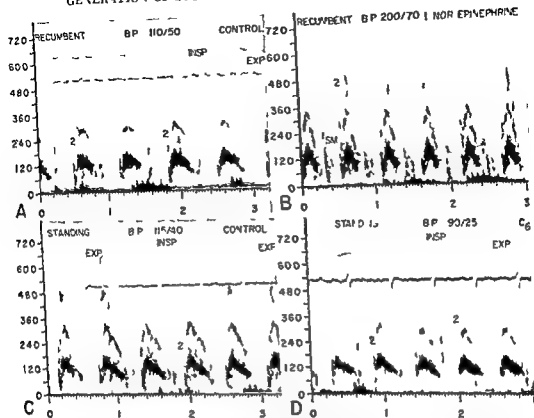


FIG 9 Change in aortic diastolic murmur with change in aortic ventricular pressure differential

Here are displayed recordings (from the same patient as in Fig 9) made at ILSD in the recumbent position before (A) and after (B) the administration of 1 nor-epinephrine intravenously and in the standing position before (C) and after (D) the administration of hexamethonium intravenously. These recordings and displays were made in as identical a manner as possible except for the differences noted. In Fig 10 (next fig) is presented a synthesis of the findings.

Both intensities (as demonstrated by the number of harmonics which are evident) and the frequency level of the harmonics are directly related to aortic pressure. The velocity of regurgitation and therefore the frequency of the murmur is raised or lowered with corresponding changes in aortic pressure. The interruption of the murmur with atrial systole is most distinct when aortic pressure is highest.

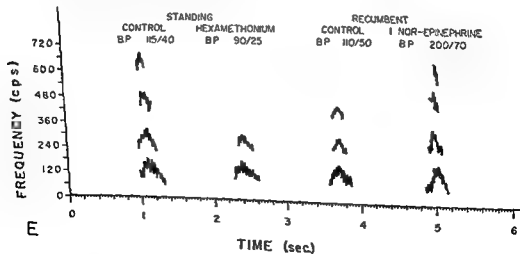


Fig 9SE

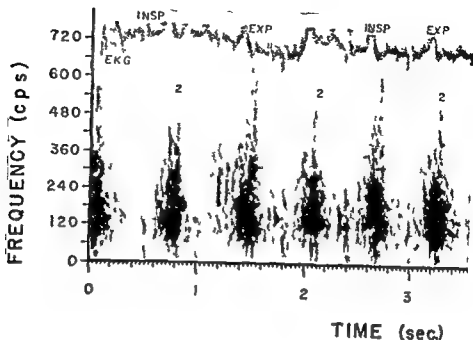


FIG. 99 Typical patent ductus arteriosus  
II SB in A. K. (757481) 2 year old female showing the typical continuous murmur built around  $S_2$

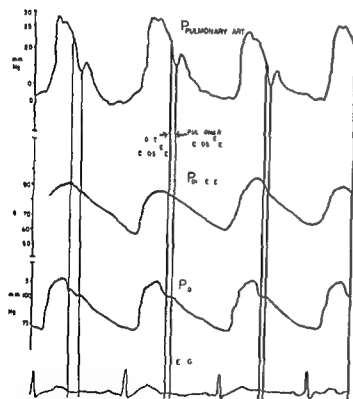


FIG. 100 Aortic pulmonary pressure differential in a normal individual

The top and lowest pressure curves are traced from actual recordings of pressure made simultaneously in the pulmonary artery and aorta in a 20 year old male with a normal cardiovascular system. The middle pressure curve is derived; it is the difference in pressure between the other two curves. In relation to the time of closure of the aortic and pulmonary valve the peak

will. This is the collision murmur (*infall geräusche*), of which Bondi (131) wrote and which is discussed earlier (p. 134).

A very important factor in determining the shape of a murmur, either its time intensity shape in the oscillogram or its time frequency shape in the spectrogram, is the time course of the pressure gradient across the orifice where the murmur is generated. The relationship between pressure gradient and shape of murmur is illustrated for the murmurs of aortic stenosis and regurgitation by the sketch in Figure 96. Figures 97 and 98 illustrate changes in the shape of the diastolic murmur when the gradient changes during the cardiac cycle or is altered by pharmacologic means.

In the case of aortic or pulmonary diastolic murmurs, I have several times observed (e.g., Figure 261) abrupt cessation of the murmur at the time corresponding to atrial systole as indicated by the P wave of the electrocardiogram. In some cases a faint transient has occurred at the termination of the murmur. This might be termed a reclosure sound. A possible explanation for the phenomenon is that ventricular pressure

occurs late in systole but not as late as one might predict on the basis of the shape of the murmur of patent ductus arteriosus. (Courtesy of Dr. Alfred E. Blum, New York City.)

is sufficiently raised by atrial systole that the aortoventricular pressure differential falls below the critical threshold necessary to maintain the murmur. Another possible explanation is that atrial systole by some mechanism influences the position of the aortic valve cusps so that competent closure is possible although it was not previously. The change which might occur with development of atrial fibrillation would shed some light on the question of whether atrial systole is indeed responsible.

A other illustration of the relation ship between

pressure gradient and shape of murmur is provided by patent ductus arteriosus. In the usual case there is a continuous murmur (Fig. 99) which has its maximal intensity and frequency span late in systole or in the vicinity of the second heart sound (see p. 400). The pressure gradient in such cases likewise demonstrates a peak in late systole (Fig. 103).

There are now available good recordings of the gradient across the mitral valve with mitral stenosis. Again the time course of the gradient bears a close parallel to the shape of the resultant



FIG. 101. Pressure curves recorded during thoracotomy in a patient with mitral stenosis. Inguineal cannulae have been used for ventricular and atrial pressure. The diastolic gradient is represented by the distance between the two curves. (Courtesy of the Orange Chapel Hall, N. C., and of the New England Journal of Medicine 71:1)

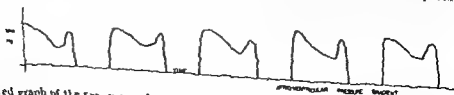


FIG. 101B. A derived graph of the pressure gradient across the mitral valve. The shape of the diastolic murmur in mitral stenosis corresponds closely to that of the curve of pressure gradient. For example compare the diastolic murmur as represented in Figure 141 and 75.



murmur (Fig 101) In cases of holodiastolic murmur from pulmonary hypertension Davidson (330A) has shown that there is an appreciable gradient between the pulmonary artery and right ventricle throughout diastole

Velocity of flow, a significant consideration from the point of view of Reynolds formula and also of jet impact in the generation of murmurs, is proportional to the square root of the pressure gradient The relation of pressure gradient to the cross sectional to the valve orifice is indicated in Gorlin's formula (p 286) The pressure gradient varies inversely with the square of the cross section of the valve In terms of the volume of flow, the size of a regurgitant orifice is probably

more important than the pressure gradient There is a direct relationship between the volume of flow and the size of the orifice, there is a square root relationship between flow and pressure gradient Gorlin (574) described studies in a patient with the combination of systemic hypertension and mitral regurgitation Reduction of systemic blood pressure to one half its pre treatment level effected only a 20 per cent reduction in the volume of mitral regurgitation In accord with the above observations is the usual failure of murmur intensity, which is probably related particularly to pressure gradient, to indicate the magnitude of regurgitant flow, which is related particularly to orifice size, in mitral and aortic regurgitation

## CHAPTER 11

# The Transmission of Sound in the Human Body

Alteration in the character of cardiovascular sound in the process of transmission to the surface of the body is inevitable. Francis Bacon (1561-1626) did a famous experiment illustrating this fact. He showed that when he put his head under water in an overturned bucket with air trapped under it speech sound as heard outside was strangely altered in character (1532). In the chest the situation is even more complex than in Bacon's experiment. There is absorption, reflection, refraction, effective attenuation and conduction at different velocities along various pathways. Clearly the transmission of murmurs determines to a considerable extent their geography, i.e., the distribution of the areas of audibility on the surface of the chest.

The following statements on the transmission of sound in the human body are based largely on empirical observations.

1 Lung is a poor conductor of cardiovascular sound.

2 Bone is an excellent conductor. The observation (589) that very loud heart murmurs audible over the chest are not affected by the inflation of a blood pressure cuff on the upper arm is probably evidence of bone conduction. Conduction of the murmur of mitral regurgitation to the scrum (1478) when the left atrium impinges on the pinc may be a similar example.

3 In general higher frequency components are attenuated more in transmission than are the lower frequency components.

4 Components at a level of frequency in the general range of the natural frequency of the thorax are best transmitted. The natural fre-

quency of the normal adult male thorax is in the vicinity of 120 c.p.s., that of the adult female thorax somewhat higher on the average and that of children's thorax yet higher (See p. 477).

5 Croedel and his collaborators (135, 600, 602, 603) found that the normal heart sounds inside the thorax have a muffled character but outside of the chest wall a metallic high pitched appearance (135). They concluded that the chest wall adds components of higher frequency to the heart sounds—that is, that part of the higher frequencies in the heart sounds are induced oscillations in the rib. The conclusion was based on observations in cases of defect of the bony thorax and on auscultating in pneumothorax and in the neck.

6 Sound, particularly murmurs, may be transmitted in the direction of the blood stream at the rate of the pressure pulse wave. The classical example is the murmur of aortic stenosis. According to the views of Hert and Harp (787) and of Lepeschkin (879) there is in effect a local reproduction of the murmur as the disturbance initiated at the aortic valve is transmitted along the blood vessel (Fig. 102 and 103). Huebner and Morris (681) noted the transmission of aortic systolic murmurs to the abdominal aorta. Aortic diastolic murmurs on the other hand were not transmitted. The Korotkoff sounds appear to be better transmitted down stream than up stream (147). Lucada (978) found that the second Duroziez murmur is transmitted downstream at about the velocity of the pulse wave. The evidence for transmission at this velocity of the pressure pulse is based mainly on timing, against the fact that the onset of the loud murmur of aortic stenosis is at

loud progressively more distant from the heart. One pitfall to avoid in such an approach being Christmas tree or diamond shaped with a peak in mid diastole, the only part detectable as one progresses away from the heart will be the more intense and progressively later portions of the murmur. However, this source of error cannot account for all the findings reported, particularly those in which the peak of the murmur is used for timing purposes. Leatham (862) published an interesting comparison (Fig 104) of the transmission of the murmurs of aortic stenosis and pulmonary stenosis to the neck. In aortic stenosis the peak is delayed suggesting a prominent mode of transmission in the wall of the aorta, innominate and carotid at a rate in the range of the pulse wave velocity. The peak of the murmur of pulmonary stenosis occurred in the neck about simultaneously with that of the murmur at the precordium, suggesting that the main mode of transmission is more directly through the tissue

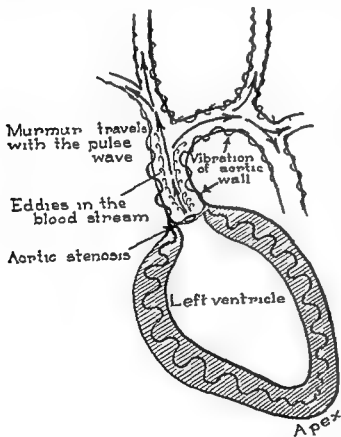


FIG 102 The transmission of the systolic murmur of aortic stenosis from the aortic valve in the direction of the blood flow and backward along the tensile contracting ventricular wall to the apex (From Kerr (745))

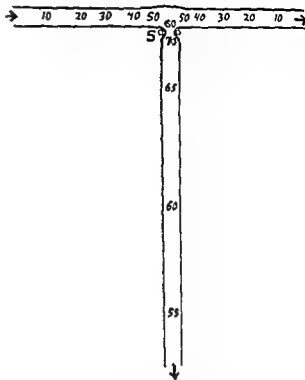


FIG 103 Loudness of murmur in decibels above hearing threshold in the vicinity of a stenosis (S) in a perfused rubber tube. Direction of flow indicated by arrows (From Lepechkin (879))

at a rate closer to that of sound in water. The last mode of transmission is less efficient (22) than the faint audibility of the murmur of pulmonary stenosis in the neck. Many times in very loud murmurs of aortic stenosis one can demonstrate that obliteration of the artery by inflation of a blood pressure cuff does not abolish and often does not even diminish the murmur in localities to the cuff on the arm (888). Possibly bone conduction is the main mode of transmission in such cases.

7 Some of the peculiarities in 'transmission' of murmurs may in fact be due to peculiar directionality of a jet which actually results in generation of a murmur in the neighbor of the area to which transmission has occurred. See page 134 for many examples. Here I will cite the systolic murmur at the right sternal border in mitral regurgitation and the aortic murmur of aortic regurgitation.

In mitral regurgitation the radiation of the murmur to the apex and axilla is possible contrary to what one might anticipate from the orientation of the regurgitant stream. The reason

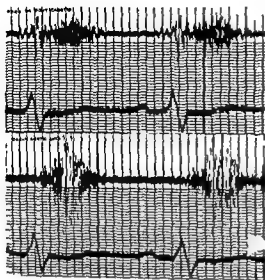


FIG 1011 Phonocardiogram of patient with congenital pulmonary stenosis showing synchronism of systolic murmur at the pulmonary area and the right carotid. The carotid tracing was taken with increased sensitivity.

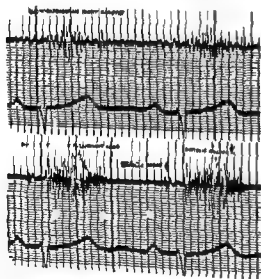


FIG 1012 Phonocardiogram of patient with rheumatic aortic stenosis. Arrows indicating delay in the peak of the systolic murmur at the right carotid artery as compared to the aortic area.

for the apical radiation has long been a matter of conjecture and to some extent experiment (148). D'Sautelle and Cray (347) thought that the insertion of the chordae tendineae and papillary muscles might determine the radiation that they might act as peculiar conductors. I am more in-

clined to the view that the fact that the left ventricle is in contact with the chest wall whereas poorly conducting lung separates the posterior aspects of the heart from the surface explains the radiation without specific reference to the papillary muscles. In 1913 I (in (911)) practiced esophageal auscultation with the particular purpose of determining whether the systolic murmur of mitral regurgitation was usually loud at this site. He found it was. If sufficiently loud any murmur might be audible in the esophagus but that of mitral regurgitation was usually the loudest. I then thought that this method permitted him to exclude mitral regurgitation as the cause of certain murmurs—particularly the late systolic murmur introduced by a systolic click (see 210). Later findings are in agreement with those of intracardiac phonocardiography. I (in (912)) and I (in (987)) report that a murmur is loudest in that chamber into which the flow of blood is occurring—e.g. the left atrium in mitral regurgitation.

In transmission to a locus distant from the area of generation a murmur may be appreciably changed in frequency pattern and therefore in approximate pitch and certainly, quality. A striking example is the murmur of aortic regurgitation which may sound quite different—in particular lower pitched—when heard at the apex and in the axilla (Fig 1013). In part this may be the result of greater attenuation of higher frequencies, in part a preferential transmission (actual accentuation) of components in the same frequency range as the natural frequency of the thorax.

There is an interesting difference in the frequency pattern of the murmurs of mitral and tricuspid stenosis. In the murmur of tricuspid stenosis one usually discovers in the SPCC components extending up to a frequency of 400 cps and occasionally as high as 800 to 900 cps—a range quite unfamiliar in mitral stenosis. Since left atrial pressure on an average attains higher levels in mitral stenosis than does right atrial pressure in tricuspid stenosis it is reasonable to assume that even higher frequencies are generated in the instance of mitral stenosis. However, these components of higher frequency in the murmur of mitral stenosis do not reach the surface of the thorax requiring as it does tran-

loci progressively more distant from the heart. One pitfall to avoid in such an approach being Christmas tree or diamond shaped with a peak in mid diastole, the only part detectable is one progresses away from the heart will be the more intense and progressively later portions of the murmur. However, this source of error cannot account for all the findings reported, particularly those in which the peak of the murmur is used for timing purposes. Leatham (862) published an interesting comparison (Fig 104) of the transmission of the murmurs of aortic stenosis and pulmonary stenosis to the neck. In aortic stenosis the peak is delayed suggesting a prominent mode of transmission in the wall of the artery, in pulmonary stenosis the peak is delayed suggesting a prominent mode of transmission in the wall of the artery, in pulmonary stenosis the peak is delayed suggesting a prominent mode of transmission in the wall of the artery, in pulmonary stenosis the peak is delayed suggesting a prominent mode of transmission in the wall of the artery.

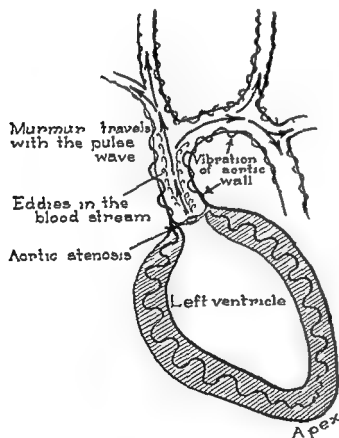


FIG 102 The transmission of the systolic murmur of aortic stenosis from the aortic valve in the direction of the blood flow and backward along the tense contracting ventricular wall to the apex (From Kerr (785))

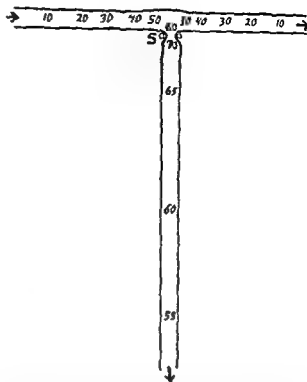


FIG 103 Loudness of murmur in decibels above hearing threshold in the vicinity of a stenosis (S) in a perfused rubber tube. Direction of flow is indicated by arrows (From Lepechkin (879))

at a rate closer to that of sound in water. The first mode of transmission is less efficient, i.e., the faint audibility of the murmur of pulmonary stenosis in the neck. Many times in very loud murmurs of aortic stenosis one can demonstrate that obliteration of the artery by inflation of a blood pressure cuff does not abolish and often does not even diminish the murmur in loci distal to the cuff on the arm (888). Possibly bone conduction is the main mode of transmission in such cases.

7. Some of the peculiarities in "transmission" of murmurs may in fact be due to peculiar directionality of a jet which actually results in generation of a murmur in the neighbor of the area to which transmission has occurred. See page 134 for many examples. Here I will cite the systolic murmur at the right sternal border in mitral regurgitation and the systolic murmur of aortic regurgitation.

In mitral regurgitation the radiation of the murmur to the apex and axilla is possible contrary to what one might anticipate from the orientation of the regurgitant stream. The reason

difficulties. Placing a speaker against the patient's back cannot be practiced as a calibrating procedure since circumferential transmission of the sound in the rib occurs rather than transmission through the chest which would be of more pertinence. Having the patient hold a small speaker in the mouth might be worthy of exploration. A vibrating signal in the esophagus would be more valid but has obvious practical drawbacks. Marked attenuation of the esophageal signal with emphasis despite relatively little attenuation of the signal applied to the back was found by Lepechkin. The esophageal signal gave results more comparable to those observed with the heart sound.

Lepechkin (879) points out that an analogy between the transmission of murmurs and the carrying of a distant sound by the wind is untenable. The velocity of sound in air is about 330 m per sec and a moderately strong wind of 10 m per sec would reduce the distance traveled by the sound per second by about 3 per cent. In the case of blood however the velocity is at the most 0.4 m per sec while the velocity of sound in water is around 1400 m per sec. This would

lead to a reduction of only 0.03 per cent in the distance traveled by the sound.

In a T tube experiment (fig. 103) Lepechkin (879) provides foundation for the view that the murmur is in effect recreated in transmission down an elastic tube. A murmur is created by a stenosis at point *A*. Arrangements are made for the outflow at *C* to be equal to the outflow at *D* therefore velocity of flow is the same in the arms *BC* and *AD* of the tube. The fact that attenuation is the same in arm *BC* and *AD* indicates no assistance to transmission by the flow of fluid. The fact that attenuation is so much greater in section *BC* than in section *AD* suggests recreation of the murmur at successive intervals down arm *AD* as the flow disturbance originating just beyond the stenosis passes down the tube.

This discussion of transmission of cardiovascular sound in the body is obviously on a descriptive and superficial plane. Von Cierke and collaborators (1407) have indicated that there are three modes of propagation of sound in the body: bulk waves, compression waves, and surface waves. There are beginnings on a comprehensive description of the physics of vibrations in living tissues.

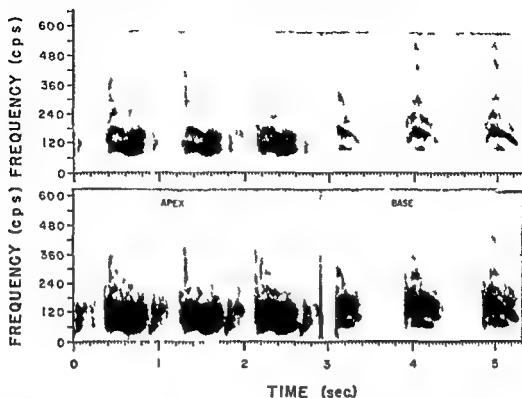


Fig. 10b. Preferential frequency transmission in murmurs.

Here are presented on the left a recording from the apex and on the right one from the base of a patient with a sea gull diastolic murmur caused by retroverted aortic cusp. Although no attempt was made to record and display the murmurs with identical gain, the relative intensity of the harmonics in the murmur at the apex as compared with those at the base (nearer the site of origin of murmur) makes it evident that the harmonics in the 120 cps range are preferentially transmitted to the apex. The patient in this case was a 45 year old male. The net natural frequency of the male thorax is in the vicinity of 120 cps.

mission through much more cardiac and other tissue than does the murmur of tricuspid stenosis, in which case the generator is immediately underlies the point of auscultation with much less intervening tissue.

The rate of transmission of cardiovascular sound in body tissues that is the transmission of any sound with frequency composition at the lower end of the acoustic spectrum (564), is not established to a completely satisfactory degree. On first thought sound might be expected to travel in the body at the same rate as sound in water (about 1400 m per sec). However as mentioned above evidence is presented by Kerr and Harp (787) that the rate of transmission is more nearly of the order of the velocity of the pulse wave (about 5 m per sec)—a striking discrepancy indeed!

Wood (1586) writes as follows:

When liquid is contained in an elastic tube, the yielding of the tube lowers the velocity of sound below the

ordinary value for sound in a given fluid. This was demonstrated by early experiments of Wertheim predicted by Helmholtz and quantitated by Sir Horace Lamb.

Although transmission of sound in the body is not strictly analogous to the situation in an elastic tube, the "yielding" of the tissues has a similar effect. Although not mentioned by Wood Stewart (see p 45) also noted the relatively slow transmission of sound in an elastic tube (1351). Lamb devised a formula for predicting the velocity of sound in an elastic tube taking into account such factors as Young's modulus of the wall material and the true velocity in the liquid.

Calibration of the intensity of heart sound and murmurs should take account of difference in the sound transmitting properties of the thorax from individual to individual. Obesity, dimensions of the rib cage, pulmonary emphysema and other factors introduce large variables. Efforts to take the factor of the chest into consideration meets

## SECTION III

### *The Auscultatory and Physical Characteristics of Major Categories of Cardiovascular Sound*





## CHAPTER 12

# The Transients

### CHARACTERISTICS OF THE HEART SOUNDS

The first heart sound is usually a duller more prolonged sound than the second. It is loudest in the tricuspid and apical areas. It can be shown to be made up of at least two components—one related to mitral closure and one to tricuspid closure usually in that sequence (Fig. 106). The duller quality of the first sound is usually represented in the SPCC by a relatively narrow frequency span with few high frequency components and relatively great intensity of the component at the lower end of the frequency scale (Fig. 107, 108, 109 and 110).

Table 6 presents information on the relative energy content at various levels of frequency of the normal first heart sound. The data are those of Williams and Dodge who published in 1926 (1966). The findings of pericardial phonocardiography are in rough agreement as are also the data of Pahlplatz (1909) who used a heterodyne analyzer which produces charts of intensity versus frequency.

The second sound is sharper in quality. It is loudest in the aortic and pulmonary areas. Before a person reaches the age of 20 or 30 years the second sound in the pulmonary area usually exceeds that in the aortic area ( $P_2 > A_2$ ) where  $A_2$  is the aortic ( $A > P$ ) is usually the case in later life. The second sound in the aortic area normally is unitary; it is related only to aortic valve closure (Fig. 106). The second sound in the pulmonary area normally is a composite of two transients related to aortic and pulmonary valve closure usually in that sequence if they are not precisely synchronous. Splitting of the second sound is likely to be heard best in the pulmonary area and elsewhere along the left sternal border. The second sound at the apex is usually only aortic closure sound.

There is a large body of evidence bearing on

the constitution of the second sound beginning at least as early as Fawcett in 1896 (41). Wiggers (1948) presented evidence for the predominantly aortic origin of  $A_2$  and  $M_2$  in the form of observations on the changes in the heart sound with various physiologic and pharmacologic maneuvers.

Various onomatopoeic devices for the heart sound of which lubb-tup (or some slight variation) is the most familiar and various other imitations of the heart sound have been invented. Palfrey (1176) imitated the heart sound by tensing a pocket handkerchief—a long strip for the first sound a shorter strip for the second (360). Larned (811) devised an ingenious method of putting one hand over one ear and by a combination of taps, strokes and scratches on the back of the hand imitating heart sounds and murmurs for teaching purposes.

The third heart sound (Figs. 111 and 112) can be detected in the majority of individuals in the first two decades of life. It is loudest (176) at LLSB and apex, in the decubitus position especially the prono-decubitus position (first after lying down) during in position following exercise of the legs with the subject in the recumbent position. All the factors enumerated favor venous return of blood. Boxer, Eckstein and Wiggers (116) found the sound in 4 per cent of normal young subjects seated but in 26 per cent recumbent. Sloan and colleagues (1397) reported that the sound was detected stethoscopically in 98 per cent of university student but could be demonstrated in 100 per cent by linear phonocardiograms and 79 per cent by logarithmic phonocardiogram. Sloan and Wi Hart (1399) found that the physiologic third sound of the dog was diminished or abolished by reduction of venous inflow to the heart.



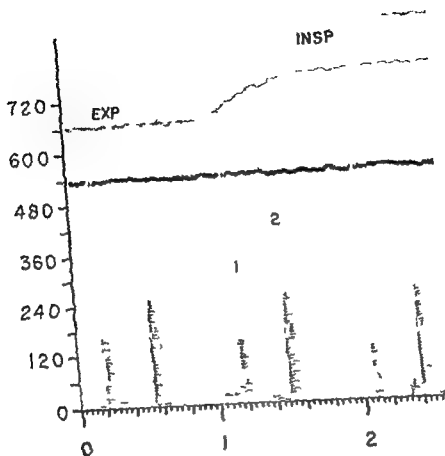


FIG. 109 Normal heart sound

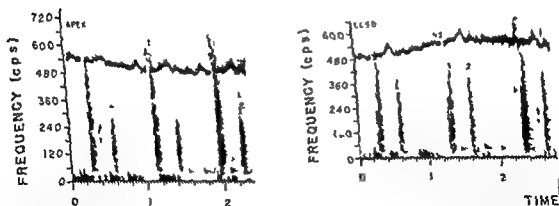


FIG. 110 Heart sound in J. J. (116.7) normal 18-year-old male. At the apex (left) only one element of  $S_1$  is demonstrated. This is presumably the mitral closure sound. It corresponds to the first of two elements of  $S_1$  which are visible at LLSB (right) and presumably represent mitral and tricuspid closure in that order. It is possible, however, that the second component of  $S_1$  shown in Fig. 110B is an arterial valve opening sound.

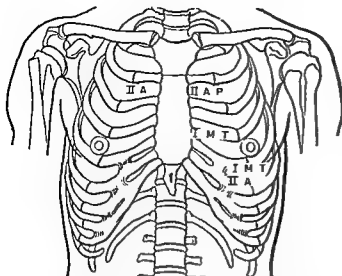


FIG 106 Constitution of normal heart sounds

Usually what is heard for second sound in the aortic area is exclusively aortic closure sound, whereas in the pulmonary area one hears aortic and pulmonary closure in that order if there is any separation of the elements. Usually in normal adults only the aortic closure sound is transmitted to the apex. The first sound appears to be a composite of mitral and tricuspid closure usually in that sequence if there is any separation. (After Leatham (850) )

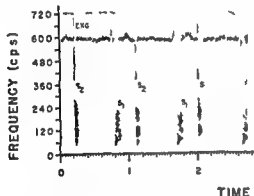


FIG 107 SPCG of heart sounds at apex in a normal 32 year old male

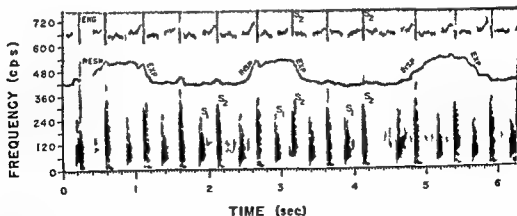


FIG 108 Pulmonary area in a normal 5 year old male. Note the splitting of  $S_2$  late in inspiration and at the beginning of expiration. On the 1 kg the time of the second heart sound is marked by a vertical line

The third heart sound is normally dull in quality and has low frequency content in the SPCG

The normal atrial, or fourth, heart sound can be detected by stethoscopy in only a minority of individuals and these in the youngest age group. It, too, is low pitched. It is usually best heard at the left sternal border. In addition to the exaggerated form of the atrial sound in presystolic gallop, atrial sounds are commonly audible in complete heart block and sometimes in atrial flutter. Atrial heart sounds are heard particularly often when complete heart block and atrial flutter are present in combination. Bennett and Kerr (87) described alteration in the intensity of the atrial sounds in a case of the latter type. In cases of complete heart block the occurrence of atrial systole independent of ventricular systole provides an opportunity of demonstrating two components of the atrial sound: the first seems to be related to tensing of the atrial wall itself and the second is probably produced in the ventricular myocardium in response to atrial systole. (One or both of the elements may have a contribution from the AV valvular mechanism.) Weitzman (1023) found that the atrial sound begins 0.12 to 0.17 sec after the onset of the P wave and 0.05 to 0.09 sec after the onset of atrial systole as signalled by the intracardiac pressure recording.

Table 7 presents Münchheimer's data (1031) on the frequency content of the four heart sounds.

Merely for sake of completeness it should be mentioned that a fifth heart sound has been described (219), located in diastole after the third heart sound but before the P wave and the

## THE TRANSIENTS

TABLE 1  
Frequency range of normal heart sounds †

| Frequency Range | First Sound |         | Second Sound |         | Third Sound |         | Atrial Sound |         |
|-----------------|-------------|---------|--------------|---------|-------------|---------|--------------|---------|
|                 | $\lambda$   | Percent | $\lambda$    | Percent | $\lambda$   | Percent | $\lambda$    | Percent |
| Below 100       | 130/130     | 100     | 130/130      | 100     | 91/130      | 69.6    | 4/130        | 3.1     |
| 50-150          | 130/130     | 100     | 135/130      | 100     | 21/130      | 16.8    | 13/130       | 10.0    |
| 100-200         | 170/130     | 93.3    | 134/135      | 99.3    | 9/130       | 1.5     | 2/130        | 1.5     |
| 150-400         | 107/135     | 5.6     | 170/130      | 93.3    | 1/130       | —       | —            | —       |
| 200-500         | 96/130      | 1.1     | 174/130      | 91.9    | 1/130       | —       | —            | —       |
| 500-1000        | 40/130      | 99.8    | 4/130        | 3.1     | 1/130       | —       | —            | —       |

The numbers and percentages listed refer to the cases with components demonstrable in the several frequency bands.

† From Mannheim (1958) table 21, p. 103.

## SPLITTING OF THE HEART SOUNDS

Normally the second sound is likely to display at least slight splitting with inspiration (Fig. 113). The basis for this is increase in venous return to the right side of the heart due to thoracic inspiration (173) and increased right ventricular stroke volume (1357). The length of ventricular systole parallels stroke volume. At the same time that venous return to the right ventricle is increased there is an influence opposite in direction and perhaps less in magnitude on venous return to the left ventricle (1061). Obviously in normal inspiratory splitting of the second sound the sequence is aortic closure sound then pulmonary closure sound. With normal continuous respiration in healthy subjects the interval between the two components of  $S_2$  usually does not exceed 0.03 sec in expiration. Boyer and Chaholm (147) point out that not only is the pulmonary component likely to be later in inspiration but the aortic component usually occurs earlier as well. See Figures 114 and 115 and Table 9. Moir and Johnson (1120) found that the same amount of stretch resulted in a greater increase in the stroke volume of the right ventricle than of the left. This result they attributed to the difference in architecture of the wall of the ventricle such that the fibers of the right ventricle are placed more nearly on a linear stretch whereas the stretch is circumferential in the case of the left ventricle. On the basis of this experi-

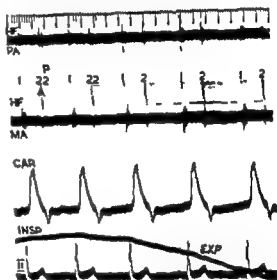


FIG. 113 Normal inspiratory splitting of  $S_2$ .

Phonocardiograms from the pulmonary and mitral areas (PA and MA) with an indirect carotid tracing (CAR) and electrocardiogram all taken simultaneously. Splitting of the second sound is confined to the pulmonary area and to inspiration (in p). The earlier component ( $A_2$ ) is shown to be caused by aortic closure by its synchrony with the diastolic notch of the carotid tracing and is transmitted to the mitral area. Later pulmonary closure ( $P_2$ ) is confined to the pulmonary area. Time intervals in the recordings shown here are 0.2 and 0.004 sec. HF refers to the fact that the characteristics of the phonocardiograph were such as to favor higher frequency components in the sounds (Courtesy of Dr. Aubrey Leatham).

Effect of respiration on QRS amplitude and the acceleration of the effects by change in right and left ventricular stroke volume have been pointed out (834).

ence in the experimental animal one might anticipate that any respiratory fluctuations in filling pressure would have a more pronounced effect on stroke volume and systolic duration in the right

TABLE 6

Frequency energy composition of heart sounds\*

| Frequency | Total Energy |
|-----------|--------------|
| c p s     | Per cent     |
| 50        | 56           |
| 60        | 27           |
| 70        | 10           |
| 80        | 4            |
| 90        | ■            |
| 100-110   | 1            |

\* From Williams and Dodge

atrial sound Culo (221) devotes five pages to a discussion of the normal fifth sound and two more to the pathologic fifth sound! We have never observed such a sound in recordings from several thousand patients. The possibility of a biventricular gallop, that is, a gallop arising in each ventricle and slightly asynchronous has not been confirmed (see p 179). It is not at all clear what the vibrations indicated by authors as a fifth sound may be.

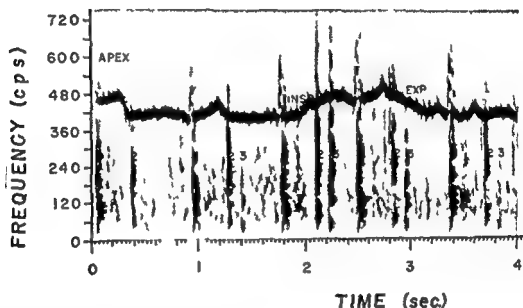


FIG. 111 In this recording from a normal 12 year old child (S.W. A96175) inspiration is accompanied by speeding of the rate (sinus arrhythmia) and accentuation of the third heart sound. There is a faint, somewhat musical systolic murmur of the type described by Still (p. 244).

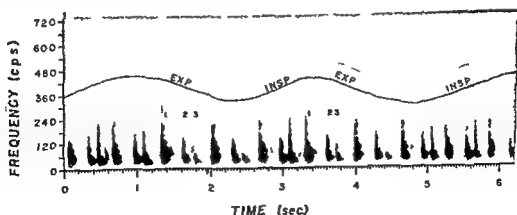


FIG. 112 Speeding of heart rate and accentuation of  $S_3$  with inspiration.

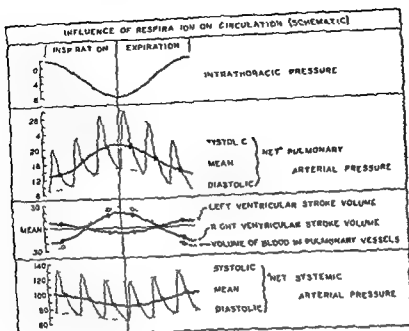


FIG 116 (From Lauson Bloomfield and Courmand (533))

of splitting of the second sound which are appropriately termed by Latham (861) *mechanical* and *electrical*.

In the *mechanical* variety of splitting (Fig 117-118) the basis is a discrepancy in the stroke volume of the two ventricles. The classical example—a side from the phenomenon of normal inspiratory splitting—is provided by atrial septal defect (Fig 110). Here because of the left-to-right shunt the right ventricle may pump considerably more blood than the left and take longer in doing so with resultant splitting of  $S_2$ .

Splitting of the second heart sound of moderate degree occurs commonly in severe mitral regurgitation (Fig 120 and 121). Presumably because of the double route of ejection from the left ventricle aortic closure occurs prematurely. That the splitting is no greater than it is may be the result of a cancelling effect of prolongation of left ventricular systole from increased stroke volume. The splitting of  $S_2$  in mitral regurgitation is likely to be evident in the pulmonary area where the systolic murmur is less audible. Slight splitting of  $S_2$  is likely to occur in ventricular septal defect for reasons identical to those in mitral regurgitation; the hemodynamics of the two situations is analogous.

Contrary to the general impression hyper-

tension of neither the pulmonary nor the systemic circuit causes conspicuous splitting of the second sound. Studies of pulmonary hypertension such as the so-called primary pulmonary hypertension have often described close splitting of the second sound (444, 1589). However the usual temporal relationship of aortic and pulmonary closure was preserved; pulmonary valve closure did not occur prematurely as a result of elevation of pulmonary artery pressure. It may be that right ventricular systole is longer in such cases. The influence of inspiration may be less impressive. Clinical experience relates splitting of  $S_2$  much more to pulmonary hypertension than to systemic hypertension. Any slight tendency to splitting would be less evident in the latter case for reasons expounded above. The clinical impression that a widely split  $P_2$  occurs with pulmonary hypertension has probably been engendered in large part by the easy audibility of the mitral opening snap in the pulmonary area of patients with pulmonary hypertension on the basis of mitral stenosis.

Contrariwise hypotension in the pulmonary artery in congenital pulmonary stenosis can result in delay of pulmonary valve closure and splitting of the second sound. Since it is the pressure differential on the two sides of the valve



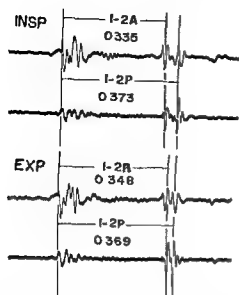


FIG. 114 Typical recording from which the measurements indicated in Figure 115 and in Table 8 were made. With inspiration I-2A is shorter and I-2P is longer than in expiration. The two recordings of each pair were made with galvanometers of different frequency response characteristics.

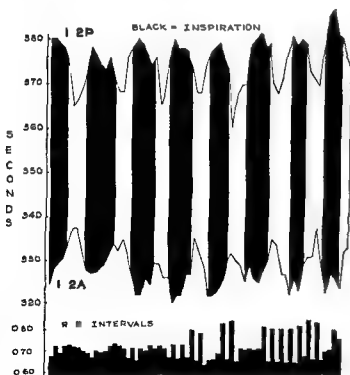


FIG. 115 Variation in splitting of S during quiet breathing in M.M. normal 21-year-old male studied in recumbent position. Because of a shifting pacemaker RR intervals were variable but demonstrated no consistent relationship to phase of respiration. The splitting of inspiration is contributed to both by delay of pulmonary closure sound and by earlier occurrence of the aortic closure sound.

TABLE 8

Changes in the second heart sound during inspiration in fifteen normal subjects\*†

|       | Mean<br>Max<br>1-2A† | Mean<br>Max<br>1-2P† | 1-2A<br>1-2P × 100 | Mean of<br>Split† | Percentage<br>of Splitting<br>Due to<br>1-2A† |
|-------|----------------------|----------------------|--------------------|-------------------|---|
| Mean  | 11.5                 | 11.9                 | 97%                | 20.8              | 49.3  |
| Range | (7-17)               | (9-24)               | (70-320)           | (11-42)           | (33-80)                                       |

\* From Boyer and Chaholm

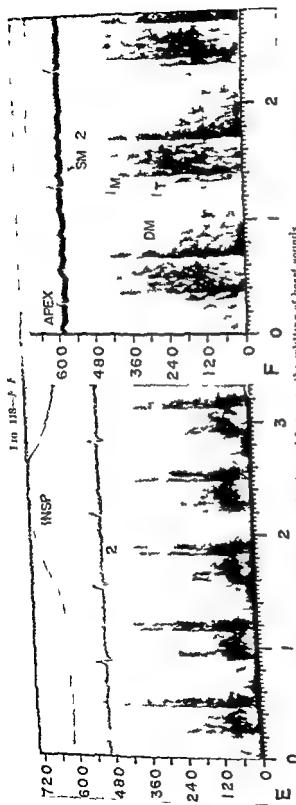
† Change (Δ) in milli seconds

ventricle than in the left. Lawson and co-workers (853) provided the lucid schematic representation of certain hemodynamic interrelations and respiration, shown in Figure 116.

In teaching students to hear splitting it is helpful to demonstrate what they should be hearing by striking two fingers on the table in a very slightly asynchronous manner. This alternating periodically with synchronous tapping of the fingers serves to convey very well the impression of respiratory splitting.

With labored respiration, with pulmonary fibrosis in which the compliance of the lung is reduced with airway obstruction, and with any other condition such that an unusually great fall of intrapleural pressure occurs with inspiration there is likely to be exaggeration of the normal inspiratory splitting of the second sound (1084). In a patient over 40 years of age without evidence of heart disease striking inspiratory splitting of S suggests some form of respiratory disease which is accompanied by exaggeration of the negativity of intrapleural pressure during inspiration. The finding is of most significance if breathing is ostensibly unlabored. Inspiratory airway obstruction as by bronchogenic carcinoma and parenchymal or pleural fibrosis are possibilities in such cases. Inspiratory splitting is a special case of the first of the two main varieties.

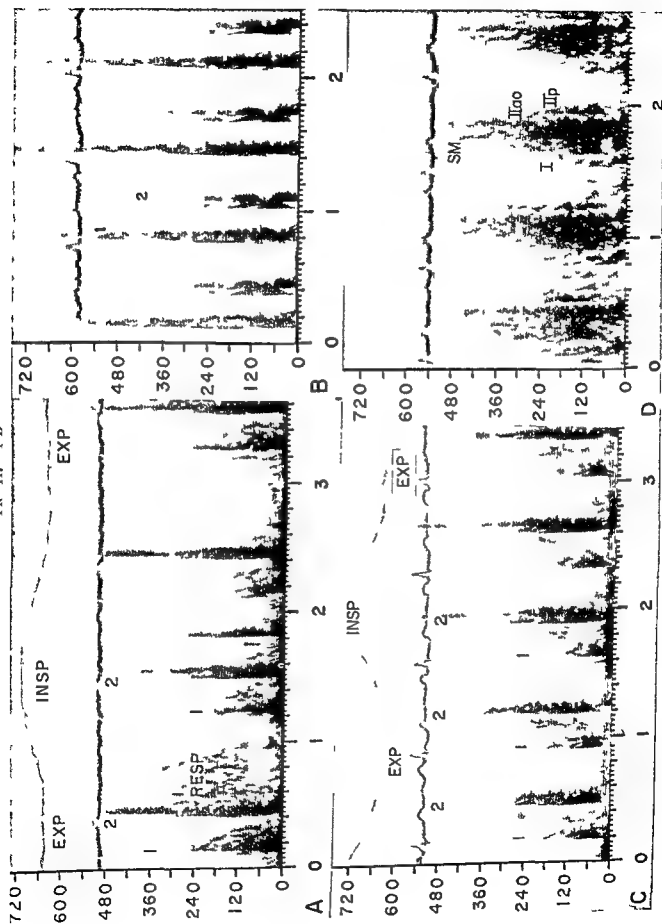
\* Another basis for splitting is synchronous closure of individual cusps of a valve as proposed by Skoda in 1854 (1395) is unlikely. No corroboration for this idea could be obtained by examining frame by frame available motion pictures of functioning heart valves made at a speed of 24 frames per second. Slight splitting seemingly demonstrated (1077) in the sounds produced by the Hufnagel plate prothesis (see ref. 1077) may be the result of a filter artifact. There is no evidence that closure followed by bouncing and reclosure occurs normally.

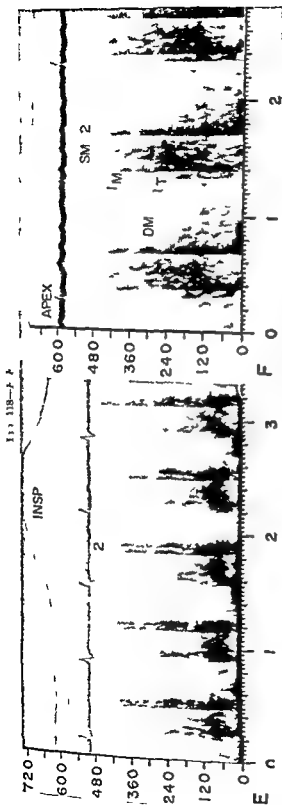


FIGS. 117 AND 118. Mechanical factors in the splitting of heart sounds.

1 pulmonary area in 13 year old child. Right ends of the murmur  $S_1$  apex in 43 year old patient with interstitial septal defect 1 arch, compo-  
nent of split  $S_2$  has frequency pattern of a valve closure and (pulmonary area in 50 year old patient with systemic arterial hypertension and  
left sided thoracoplegic for pulmonary tuberculosis) Split 1 2 for dual is caused by evoked negativity of intrapulmonary pressure  $S_1$  critically and  
or late systolic murmur believed to be extraneous. D pulmonary area in pulmonary artery stenosis is probably similar to 1 note. Christmas tree effect  
in a stenotic murmur and delayed pulmonary closure sound or pulmonary reversed note (116). The systolic murmur in 11 year old continues slightly  
after the aortic closure (116).  $S_2$  pulmonary area in 20 year old patient with mitral regurgitation. There is splitting of the  $S_2$  in 1 sound which  
is exaggerated by inspiration. Note the decrease in systolic murmur  $S_1$  apex in a patient with rheumatic mitral regurgitation. The systolic murmur  
begins immediately with the second component of the plot first sound which therefore in part is unitary—prevalent is only aortic closure sound 1—  
delayed and has an anomalous relation to the frequency sound. The second sound is unitary—prevalent is only aortic closure sound 1—  
and is followed immediately by a decrease in the total murmur which probably had its origin at the aortic orifice because of the aortic regurgitation  
after it is possible the second component of  $S_2$  is an aortic ejection sound which is usually well heard at the apex.

FIG. 117—(A-D)





**Fig. 117 and 119** Mechanical factors in the splitting of heart aorta

[illegible]

that controls its closure, the effect of pulmonary hypotension is relatively greater by reason of the ventricular hypertension. When ventricular systolic pressure is in the range of 100 to 200 mm Hg, it might be anticipated that a longer time will be taken for intraventricular pressure to fall below that in the pulmonary artery.

In a sizeable group of cases of pure pulmonary stenosis, Leatham and Weitzman (866) found a

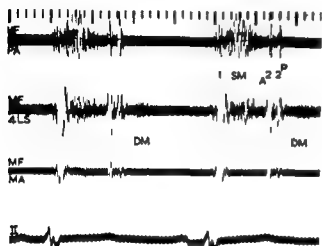


FIG 110 Split  $S_2$  with ASD

The pulmonary closure sound is louder in the pulmonary area; the aortic closure sound is louder at the apex. There is a systolic ejection murmur resulting from high flow in the right ventricular outflow path and stopping in a characteristic fashion before  $S_2$ . Incomplete right bundle branch block is suggested by the form of the QRS in the electrocardiogram. (Courtesy Dr Aubrey Leatham)

direct correlation between the level of right ventricular pressure and the degree of separation of the aortic and pulmonary components of  $S_2$ . Although the difference between ventricular and pulmonary pressure is a simple and rather obvious basis for the splitting, the possibility of a slower and more prolonged right ventricular systole as a contributing factor is also present.

Gray (587) in writing about paradoxical splitting of the second heart sound—that is, aortic component following pulmonary to produce splitting which was observed mainly in expiration—stated that 10 of 23 cases of aortic stenosis and 10 of 29 cases of patent ductus arteriosus showed the phenomenon. In the latter group increased stroke volume of the left ventricle

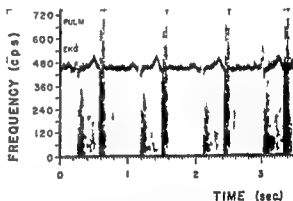


FIG 120 Split  $S_2$  with mitral regurgitation

I. M. (764492) 10 year old male had at the apex typical auscultatory findings of MR.  $S_2$  consistently split

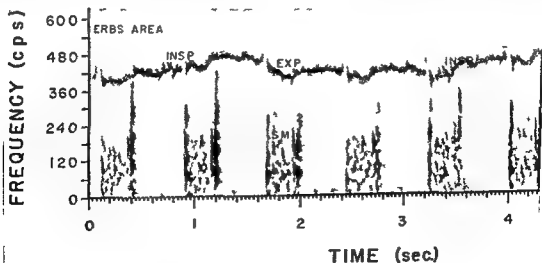


FIG 121 Split  $S_2$  of mitral regurgitation

Erb's area in J. Y. (763674) 16 year old female with rheumatic mitral regurgitation and moderate pulmonary hypertension. An early systolic click is seen in some cycles.  $S_2$  is fairly consistently split; the pulmonary component is clearly second. The splitting is slightly increased with inspiration. (See Fig 55 for recording at apex)

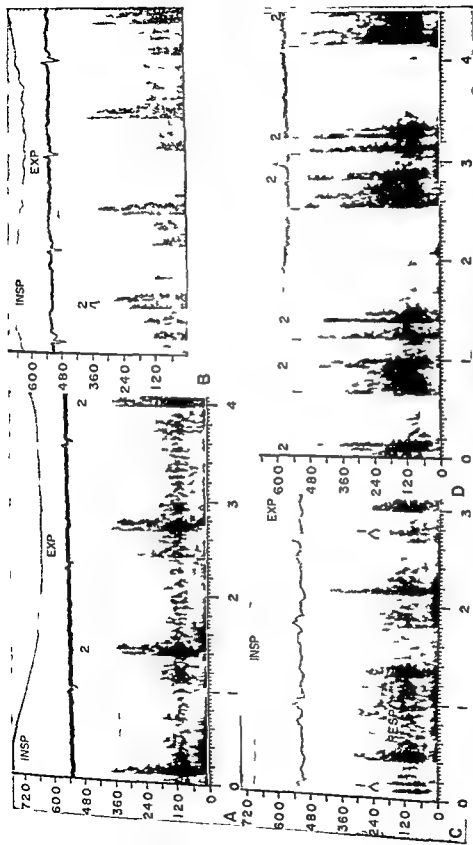


FIGURE 1. Left bundle branch block, note 1 is aortic effect of expiration. B pulmonary area, right bundle branch block, note exaggeration of

splitting with inspiration. C plot first sound in left bundle branch block. Although not clearly split the second sound in expiration. D mild regurgitation second sound split in normal cycles both first and second sound split with ventricular premature contractions note the decreased aortic murmur which is held by the

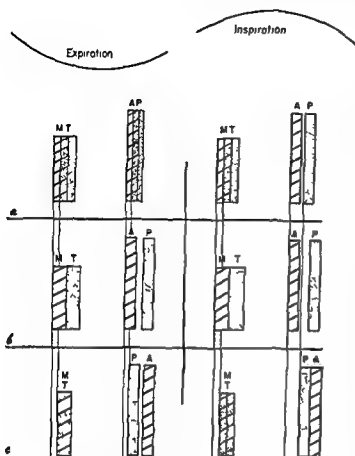


FIG. 123 Effect of respiration on the splitting of heart sounds in bundle branch block.

(a) Normal (b) right bundle branch block and (c) left bundle branch block. Note that with neither type of block is splitting of the first sound to be expected and that splitting of the second sound in left bundle branch block is paradoxical and of lesser degree than in right bundle branch block.

over the right is the responsible factor. The splitting in aortic stenosis has a basis comparable to that in pulmonary stenosis.

In the electrical variety of splitting of  $S_1$  (Fig. 122) the basis is a slower spread of the triggering impulse in one ventricle as compared with the other. The classical example is bundle branch block. Particularly in right bundle branch block is there likely to be impressive splitting of  $S_1$  since the normal tendency for pulmonary closure to lag behind aortic closure is exaggerated by the delay in triggering of the right ventricle. The delay in pulmonary valve closure is further exaggerated in inspiration through an additive effect of normal inspiratory splitting.

In left bundle branch block the degree of splitting is likely to be less impressive because the

delay in aortic valve closure tends merely to cancel out the normal slight lag in pulmonary valve closure. Furthermore, with inspiration the splitting is likely to be least, in left bundle branch block the splitting of  $S_1$  is minimal in expiration. The reason will be evident after a bit of thought. The different effects of inspiration on splitting in the two varieties of bundle branch block are graphically represented in Figure 123 and provide a bedside method for distinguishing the two forms.

In both right and left bundle branch block as well as in mitral regurgitation the splitting of the second sounds tends to become fixed when heart failure develops (Fig. 124), that is, respiration does not have its usual effects (1218a).

True splitting of  $S_2$  is fairly uncommon in bundle branch block, contrary to the conclusion of King and McFadden (795) who reported it in 28 of 56 cases in a stethoscopic study. The frequent association of presystolic gallop and bundle branch block may be responsible for a mistaken impression of the incidence of split  $S_2$  in bundle branch block (897). Contro and Lissada (286) found "three groups of vibrations" in the first sound in two cases of left bundle branch block. The last group of vibrations in the case they picture is so late as to suggest a great vessel snap ("ejection sound") rather than a true part of  $S_1$ . True splitting of  $S_2$  is more likely to occur in congenital bundle branch block as in isolated abnormality (Fig. 125). This finding accords well with the finding of Braunwald and his colleagues (168) that only in this type of case is there significant asynchrony in onset of right and left ventricular systole.

Conditions which may simulate splitting of  $S_2$  (Fig. 126) are (1) presystolic gallop (919) and (2) the early systolic click of disease of the great vessels (see below). Splitting of  $S_2$  may be simulated by a late systolic click of pericardial origin (preceding  $S_2$ ) and by a mitral opening snap or early diastolic snap of constrictive pericarditis or a protodiastolic gallop (all following  $S_2$ ). Protodiastolic gallop is likely to endow the rhythm with a evidence rarely encountered with true splitting. However the sound of constrictive pericarditis since it is located closer to the second sound is more likely to confuse

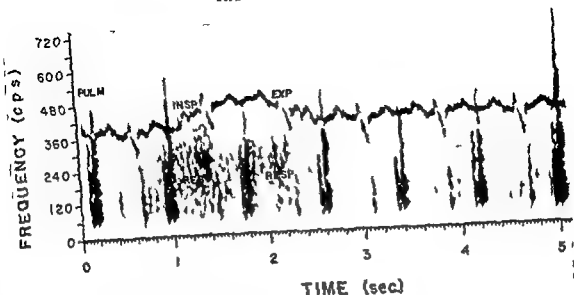


FIG 11 Left bundle branch block without respiratory variation

J D T (6 yos) 7 years old has idiopathic myocarditis 10 mls because of incompetence of the heart no exaggeration of the splitting occurs with inspiration

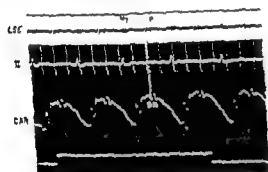


FIG 12 Congenital right bundle branch block

Exaggerated asynchrony of the two components of the first and second sounds resulting in abnormally wide splitting With inspiration upward deflection of the baseline there is further delay in pulmonary valve closure and further widening of the second sound (note of Harvey and Terloff (65) and of Cretschmer)

Because of much overlap of the degree of splitting with that occurring normally splitting is of less diagnostic value in the pathologic states with which it may occur atrial septal defect bundle branch block mitral regurgitation and respiratory disease resulting in exaggerated cyclical variations in intrapleural pressure splitting has more pathologic significance (1) when it occurs in adults than when it occurs in children (2) when splitting is exaggerated with expiration rather than with inspiration (suggesting

the paradoxical splitting of left bundle branch block aortic stenosis or patent ductus arteriosus (3) when there is no clinical evidence of respiratory disease or labored respirations and (4) when splitting persists unaltered throughout all phases of respiration (usually the case in ASD) so-called 'fixed splitting'

#### FACTORS AFFECTING THE HEART SOUNDS

Obviously the heart sounds are louder when the chest wall is thin and fainter when it is thick or when there is pulmonary emphysema or obesity. Anatomical changes—in the size of the chambers of the heart and great vessels or in the rotation of the heart—by altering the relation of the various portions of the heart to the chest wall cause change in the heart sounds. For example idiopathic dilatation of the pulmonary artery is likely to be accompanied by a loud  $P_2$  even though there is no pulmonary hypertension. In a similar reaction  $P_2$  is almost always accentuated in cases of atrial septal defect even when pulmonary hypertension is absent. Enlargement of the right ventricle with atrial septal defect is likely to result in the right ventricle's representing the cardiac apex. As a result the pulmonary closure sound is likely to be heard at the apex



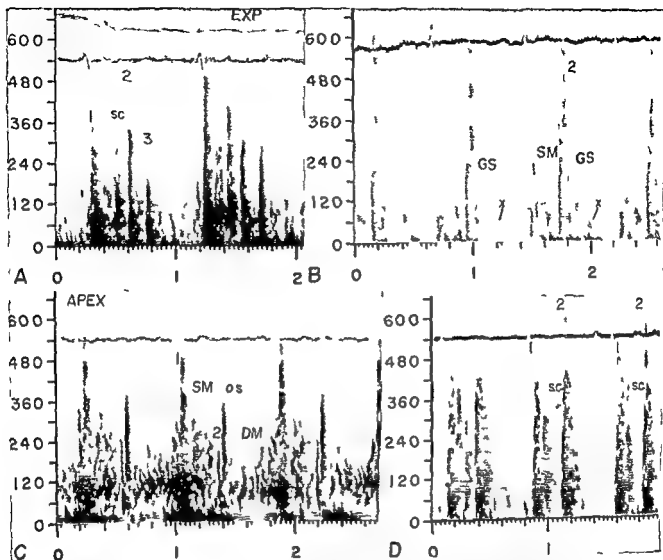


FIG 176 Phenomena simulating split sounds

A late systolic click of about the same intensity is that seen at the apex was also present at left lateral border and pulmonary area. There is a history of acute rheumatic fever during which pericarditis was observed four months before the recording. Third heart sound is also present. B This patient with mitral stenosis has considerable pulmonary hypertension with resultant accentuation of the pulmonary closure sound. The temporal relationship of the two components of  $P_2$  is not altered. There is a faint sound in mid diastole (which is difficult to identify) at the termination of the Graham Steell murmur. It is too early for an atrial heart sound and perhaps too late for a third heart sound. It may represent a valve closure sound—light leak occur through a small separation of the cusp as long as the pressure differential is sufficient to maintain the separation. As soon as the pressure gradient drops below a certain threshold coaptation of the previously separated surface occurs with production of an audible sound. C Recording at the apex in a patient with severe mitral stenosis and a moderate degree of mitral regurgitation. Note the snapping mitral closure sound, the systolic murmur and the diastolic murmur with presystolic crescendo. There is seemingly a split second sound. Actually it is clear that this is in fact a second sound followed closely by an opening snap. This patient also has aortic stenosis. The aortic closure sound is so attenuated that it is doubtful that it is transmitted to the apex where the pulmonary closure sound alone is represented. That the sound marked OS is indeed mitral opening snap is corroborated by its snapping appearance, pure frequency content and elevated frequency bottom. D An example of early systolic sound in the pulmonary area caused by dilated pulmonary artery due in this case to atrial septal defect. In addition both the first and the second sounds are lightly split.

as is also the diastolic rumble of relative tricuspid stenosis.

Aside from these extravascular and extracardiac factors affecting the heart sounds there

are valvular factors which can be classified as follows:

- 1 The speed of valvular closure
- 2 The force of valvular closure

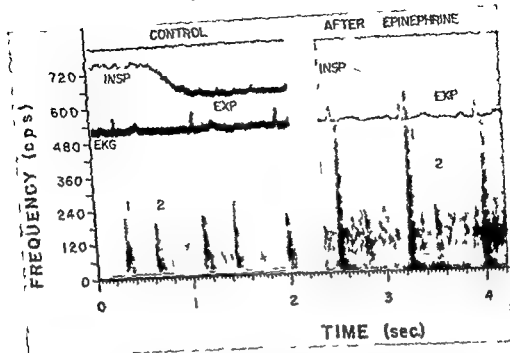


FIG. 175 Effect of epinephrine on normal heart sounds

Demonstrated in 4 are the heart sounds recorded at the lower left sternal border in a normal subject. In B is a recording from the same area made under identical condition of amplification five minutes after the administration of 10 cc of epinephrine 1:1000 subcutaneous. The second heart sound is little changed (systemic blood pressure was 100/70 mm Hg during the control recording and 114/61 mm Hg after epinephrine). The striking change in the first sound consists of increase in peak frequency (frequency pin) appearance of more conspicuous harmonics and increase in overall loudness as indicated by blackness. The first two features account for the impression of "snappiness" which the first sound after epinephrine conveys to the ear.

The accentuation of the first sound is probably closely related to the subjective palpitation which accompanies epinephrine administration. Increase in the velocity of valve closure is probably principally responsible for the accentuation. As is demonstrated in B, higher frequencies are produced and it is largely the contributions of these to total intensity which are responsible for the overall intensification.

Excessive background noise appeared in the recording after administration of epinephrine. The amplification during the act of recording and analyzing was identical in the two cases. Several explanations for the increased background noise in the second recording are possible: (1) increase in ambient (room) noise; (2) muscle noise from the tension engendered by the drug; (3) vascular noise from circulatory changes.

### 3 The excursion in valvular closure

#### 4 The physical quality of the valve

The faster a valve closes the louder is likely to be the sound produced. The youthful heart displays snappy heart sounds for this reason. Further more administration of adrenaline in doses which produce little or no change in intravascular pressures may abbreviate isometric contraction<sup>2</sup> (40) and accentuate the first heart sound (Fig

Strictly speaking it is not abbreviation of isometric contraction which is operating in producing the accentuation of the first sound into the A<sub>1</sub> valve are already closed during this period. However there is usually a direct relation between the velocity with which they are closed and the duration of isometric contraction.

127) The loud first heart sound in thyrotoxicosis probably has the same basis. Many of the manifestations of thyrotoxicosis appear to be the result of potentiation of the effect of catecholamines by thyroid hormone. Considered in myocardial disease the first sound may be described as dull of poor quality. In this situation even though intravascular pressures are normal the lower rise in intraventricular pressure makes for a dull sound. Not only is the sound produced by rapid closure of a valve louder but it also has higher frequency content according to a general principle defined as follows by Lamb (837) the higher harmonics are excited in greater

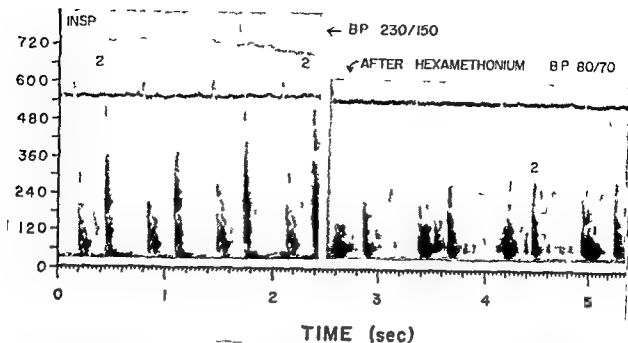


FIG. 128 Heart sounds before and after administration of hexamethonium to a hypertensive patient

In this pair of tracings from the left lower sternal border variations in blood pressure were accompanied by little change in the first heart sound which in each instance is split possibly into mitral and tricuspid components. At the hypertensive level the second sound is louder (blackier) and has a greater frequency span. The variable involved here is *force of closure* (velocity of closure, an important factor in the accentuation of the first sound (S<sub>1</sub>)) in Figure 127 is intimately related to force of closure. It is likely that aortic valve closure is more rapid at elevated levels of diastolic pressure.

*Electrical interference at 240 cycles is demonstrated*

relative intensity the more abrupt the character of the originating disturbance.

Accentuation of the second heart sound with arterial hypertension of the systemic or pulmonary circuit is familiar and represents a change related to increase in the *force* or *pressure* of *valvular closure*. (See Figure 128 for demonstration of the reversal of this effect.) Because of the double origin of P', the second sound in the pulmonary area may be louder than that in the aortic area in cases of systemic arterial hypertension, especially in young persons. The reason for this is that the aortic component of the second sound has been so accentuated that the combination of closure sounds is louder than the aortic closure sound alone as heard in the aortic area.

The first heart sound may be accentuated in association with hypertension of either circuit. In this situation increased force cannot be the factor in the accentuation since the valve closes when pressure in the ventricle exceeds that in the atrium and atrial pressure may not be elevated. The factor responsible for the accentuation must

be increased rapidity of closure. If the phase of ventricular contraction preceding opening of the arterial valves is not prolonged then to attain the level of diastolic pressure in one or the other of the great vessels the pressure in the ventricle must rise faster than it does normally.

In so-called "auscultatory alternans" which sometimes accompanies pulsus alternans (p. 421) the alternately stronger and weaker heart sounds are dependent on differences in the force of valve closure.

The excursion of the valve during closure is another important factor in the intensity of the first sound at least and possibly of the second. It has already been pointed out on page 112 that the intensity of the first heart sound in various grades of atrioventricular dissociation (Fig. 129) finds interpretation in the influence of atrial systole on the degree of separation of the AV valve cusps and the extent to which the belly of the cusps is displaced toward the atrium. The first heart sound is likely to be accentuated in ventricular premature contractions due to the

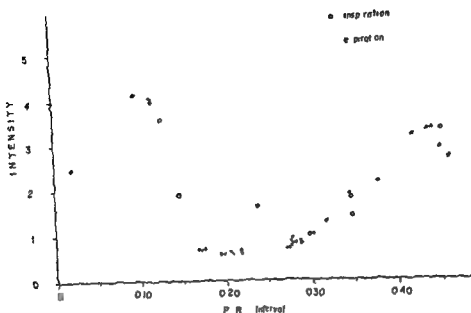


FIG. 120. The relation of intensity of the first heart sound (in arbitrary units) to the duration of the P-R interval (in seconds) in a dog in which complete heart block was produced surgically by the method of Starzl and Cretner (1934). The intensity of the first sound is presumed to be related directly to the degree of separation of the cusps at the time of contraction of the ventricle. With short P-R intervals one can infer wide separation of the cusps (or deep displacement of the cusp bellies) caused by atrial systole. At longer P-R intervals (in the normal range) the cusps would appear to be close together, probably caused by the "leaking of the jet" phenomenon described by Henderson and Johnson (1930). With still longer P-R intervals there has been time for the cusps to return to a more wide open, more neutral position. Similar curves in children have been reported (1932) (Expiration includes readings in both the expiratory phase proper and the phase of expiratory apnea) (Observation of Dr. S. H. Boerlin).

occurrence of valvular closure during early diastolic filling when the valve is relatively wide open. The tricuspid closure sound in atrial septal defect is likely to be accentuated (see p. 349) probably because of a wide open position at the onset of ventricular systole as a result of long continuing ventricular filling. In part the accentuated first sound in mitral stenosis may be due to displacement of the bellies of the mitral cusps far into the ventricle because of elevated pressure in the left atrium at the time that the ventricle contracts. The short excursion of the aortic leaflets in aortic stenosis is a factor which tends to produce attenuation of the second sound.

In this discussion of the relation of valve position to intensity of the heart sound it will be noted that the current thought is that events at the cusp belly rather than at the coapting margin are important in the production of the sounds. Therefore when it is stated that the valve is wide open or almost closed it is really the position of the cusp belly that is being referred to. Movement of the margin of the cusps of the AV valves may in fact be minimal (1936).

Fibrosis and calcification tend to accentuate it. With extracardiac aortic second sound may occur largely because the cusps of the aortic valve are spread minimally. The fact that diastolic pressure is lower is also a factor, of course. With a large stroke volume it is possible that Rousslet predicted from his observation in a model (p. 40) accentuation of S<sub>2</sub> may occur from wide separation of the cusps of the aortic valve. Other clinical examples of this mechanism may be the accentuated pulmonary closure sound in atrial septal defect and the accentuated aortic closure sound in patent ductus arteriosus.

A faint first sound is a usual accompaniment of P-R interval longer than 0.20 sec. as in the first degree heart block of active rheumatic carditis (1885, p. 123). The intensity of S<sub>1</sub> has been recommended as a useful clinical sign for following the progress of patients with rheumatic fever. The finding is thought to have its basis in a semi-closed position of the AV valves because of a

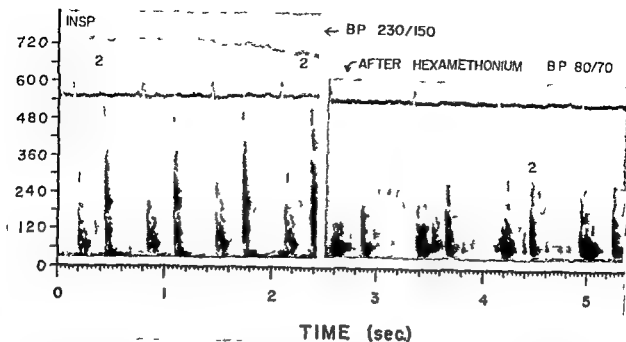


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Electrical interference at 240 cycles is demonstrated.

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## THE TRANSIENT

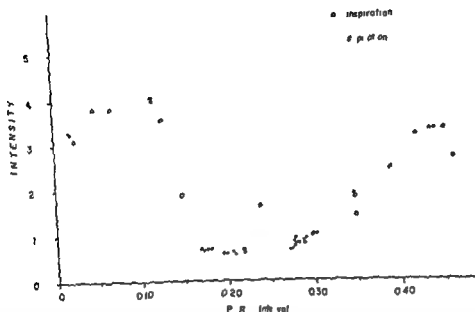


FIG. 139. The relation of intensity of the first heart sound (in arbitrary units) to the duration of the PR interval (in seconds) in a dog in which complete heart block was produced surgically by the method of Sural and Coertner (1934). The intensity of the first sound is presumed to be related directly to the degree of separation of the cu p at the time of contraction of the ventricle. With short PR interval, one can infer wide separation of the cu p (or deep in placement of the cu p bellies) caused by atrial systole. At longer PR intervals (in the normal range) the cu p would appear to be close together, probably caused by the breaking of the jet phenomenon in diastole by Henderson and Johnson (1935). With still longer PR intervals there has been time for the cu p to return to a more wide open, more neutral position. Similar curves in children have been reported (1382). Expiration includes respiration in both the expiratory phase proper and the phase of expiratory apnea (Observations of Dr. S. H. Boyer M.D.)

occurrence of valvular closure during early diastolic filling when the valve is relatively wide open. The tricuspid closure sound in atrial septal defect is likely to be accentuated (see p. 349) probably because of a wide open position at the onset of ventricular systole as a result of long continuing ventricular filling. In part the accentuated first sound in mitral stenosis may be due to displacement of the bellies of the mitral cu p far into the ventricle because of elevated pressure in the left atrium at the time that the ventricle contracts. The short excursion of the aortic leaflets in aortic stenosis is a factor which tends to produce attenuation of the second sound.

In this discussion of the relation of valve position to intensity of the heart sounds it will be noted that the current thought is that events at the cusps chiefly rather than at the closing margins are important in the production of the sound. Therefore when it is stated that the valve is wide open or almost closed it is really the position of the cu p bellies that is being referred to. Movement of the margin of the cu p of the AV valves may in fact be minimal (1376).

Fibrosis and calcification tend to accentuate it. With extrasystoles a faint second sound may occur largely because the cu p of the arterial valves are moved minimally. The fact that diastolic pressure is lower is also a factor of course. With a large stroke volume it is possible that as Bourneau pointed out from his observations in a model (p. 40) accentuation of S may occur from wide separation of the cu p of the arterial valves. Other clinical examples of this mechanism may be the accentuated pulmonary closure sound in atrial septal defect and the accentuated aortic closure sound in patent ductus arteriosus.

A faint first sound is a usual accompaniment of PR intervals longer than 0.20 sec. as in the first degree heart block of active rheumatic carditis (1888, p. 123). The intensity of S<sub>1</sub> has been recommended as a useful clinical sign for following the progress of patients with rheumatic fever. The finding is thought to have its basis in a semi-closed position of the AV valves because of a

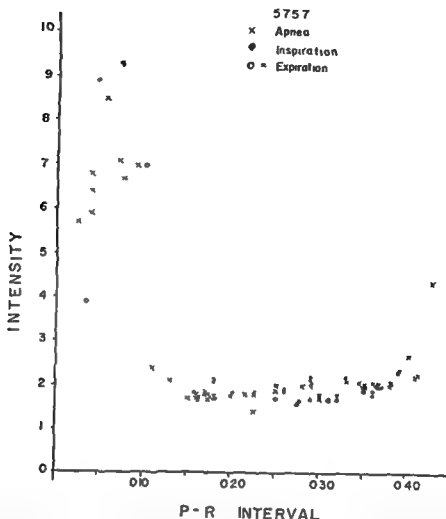


FIG 120B Relation of intensity of  $S_1$  (in arbitrary units) to the P-R interval in another dog with surgically induced complete heart block. With very short I-R intervals (below 0.05 sec) and longer P-R intervals (over 0.12 sec)  $S_1$  was least loud. A secondary increase in  $S_1$  intensity at longer P-R intervals was less impressive than in the dog shown in Figure 120A (Observations of Dr S H Boyer IV)

pulling in of the valve curtains in the wake of the atrial ejection jet

A fourth important factor in the intensity of the heart sounds is the physical state of the cusps. This influence is seen most impressively in the instance of the accentuated sounds that occur with fibrosis of the valves. A wooden snapping or clicking sound is likely to result. The snapping character of the first heart sound in mitral stenosis probably results mainly from the fibrosis in the mitral leaflets and chordae tendineae. An early sign of rheumatic affection of the aortic valve is accentuation of the aortic second sound (Figs 130 and 131). Changes in the valve cusps are responsible for the ringing and often somewhat although not necessarily, accentuated (intensified) "A<sub>2</sub>" of luetic aortitis, referred to by Potain as *bruit de tabourka*. In the past (419, 739, 1112)

a ringing A<sub>2</sub> in a normotensive individual has been accepted as *prima facie* evidence of syphilitic aortitis. The intimal atherosclerotic changes which occur secondary to the medial change and are responsible for the demonstration of calcification in the sinuses of Valsalva radiologically (874, 1055) are probably largely responsible for the change in the second sound. However, another factor which is with that just mentioned not mutually exclusive may be wider separation of the cusps when open because of dilatation in the region of the aortic ring (a special case of factor three above).

Ringings are a quality of the heart sounds which corresponds to musicality in murmurs. Ringing of heart sounds, as in the case of the second sound in arterial hypertension and the first sound in mitral stenosis is represented in the spectrogram

by harmonic 'knobbing' (e.g. Fig. 289). This corresponds to the harmonic banding seen in mitral murmur. A valve closure sound which is accentuated on the basis of fibrosis and calcification

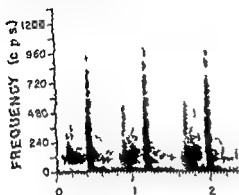


Fig. 130. Aortic stenosis with valvular fibrosis.

Aortic stenosis in a normal 15-year-old girl (S.B., 1972) with early aortic valvulitis in a rheumatic basis. The second aortic sound (S<sub>2</sub>) is very ringing in character with harmonic pattern and accentuation of both intensity and frequency span. These changes probably are caused by valvular fibrosis. There is an early diastolic murmur (D<sub>2</sub>) (S<sub>2</sub>) probably a protosystolic click and (S<sub>2</sub>) (S<sub>2</sub>) early aortic stenosis.

of the valve may have a wide frequency range with relatively little exaggeration of the intensity of the low frequency components. When the accentuation is due to increased force of closure there is an equally wide frequency range represented however the low frequency component are greatly exaggerated.

The high first sound of mitral stenosis has already been discussed. The first sound in mitral regurgitation is dull forceful tenor, of the valve is probably impossible because of the leak. In aortic and pulmonary regurgitation the second sound, even that part contributed by the valve which is diseased is usually well preserved and often is even accentuated because of the fibrotic change in the valve and possibly because of more rapid closure of the valve. Often, the valve closure sound appear to be prolonged directly into the murmur. As can be deduced from the discussion above, an interplay of factors in aortic stenosis may result in a low, increased normal or reduced depending on which factor predominates.

In the chapter on the generation of sound in the cardiovascular system the view will be

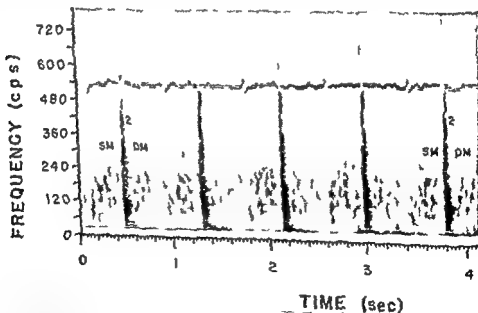


Fig. 131. Rheumatic aortic valvular disease in normal 15-year-old patient.

There is a systolic murmur produced probably by a moderate degree of aortic stenosis. This murmur has a peak of intensity and frequency in mid-systole. The second sound is followed more laterally by a decrescendo diastolic murmur. The pulse pressure in this patient is not as low as the patient is in general asymptomatic. The greatly accentuated second sound (S<sub>2</sub>) is noteworthy. There is a wide frequency span and intensification of the sound throughout its normal frequency range. The intensification of S<sub>2</sub> in this instance is caused by fibrosis of the valve. Such a firm valve would be expected to produce a higher closure



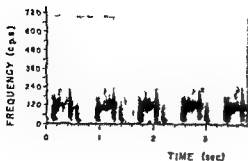


FIG. 132 Protodiastolic gallop

Recorded at apex in I C (585527) 17 years old recovering from rheumatic fever. He has mitral regurgitation (note the systolic murmur SM); the protodiastolic gallop C is the accentuated  $S_2$  which usually accompanies this valve lesion.

closure is brought to a more abrupt halt than normally is the case.

The multiplicity of the factors influencing the intensity of the heart sounds makes it unlikely that cardiac output can be estimated thereby (472, 473, 474).

### DIASTOLIC CATOPS

Two main varieties of diastolic gallops<sup>4</sup> are identified: (1) *protodiastolic* (ventricular, rapid filling, (498) or third sound) gallop (figs. 132 and 133), which is essentially an accentuation of the normal third heart sound, and (2) *presystolic* (atrial, or fourth sound) gallop (figs. 133-135).

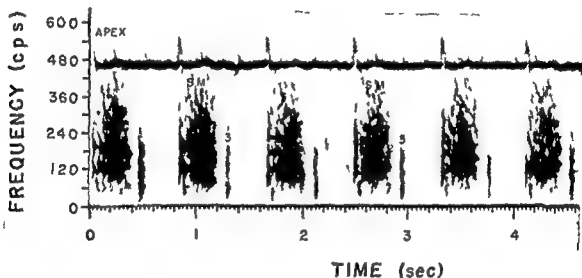


FIG. 133 Protodiastolic gallop with mitral regurgitation

Apex in I 1 (179814) 17 year old female who has rheumatic heart disease with mitral regurgitation producing a holosystolic murmur and accentuation of  $S_2$ .

pressed that the heart sounds are valve closure sounds and that they are produced not so much by collision of the coapting margins or even by snapping of the valve curtains but rather by the generation of pressure transients when local flow responsible for valve closure is brought to an abrupt halt by the inextensible collagenous valve when its limit of stretch is attained. The above analysis of factors altering the character of the heart sounds does not uncover any phenomenon inconsistent with this theory. In the case of the ringing sounds which accompany organic change in the valve it is possible that a very slight yielding (of which the normal valve structure is capable) is eliminated so that local flow of valve

and 136), which is essentially an accentuation of the normal fourth heart sound. Both may be present in a given patient (fig. 137). Two other varieties of gallop are variations on these basic: two *summation gallop* (fig. 138) occurs when both types of gallop are present and the heart rate is so rapid that they occur synchronously or in immediate succession. *Mesodiastolic gallop* is the term employed when the rate is so rapid

<sup>4</sup> Because of the difficulties in distinguishing a gallop which has come to connote a pathologic state in all instances from its normal counterpart some such as William Evans of London (439)—have favored use of the expression *triple rhythm*. This has the advantage of not prejudicing the decision of what its significance is.

that the gallop would occur in the middle of diastole and it is impossible to tell whether it is fundamentally a protodiastolic or a presystolic or possibly a unimodal gallop. The term quadruple rhythm or train rhythm is used when both types of gallop are present without summation (974).

In a more clinical chart and in deciding whether a given diastolic sound should be called a gallop or a physiologic sound (S<sub>3</sub>) or S<sub>4</sub> are known by the company they keep. Depending on the age of the patient and the state of the heart as evidenced by other signs, one or the other designation is assigned. Warren writes as follows (111): "A knowledge is required perhaps we can avoid the confusing Dr. Jehu Mr. Hyde, a term of nomenclature that results in naming the same sound differently as either a heart sound or gallop depending primarily on the company it keeps. Actually all evidence seems to be in agreement with the statement made above that diastolic gallop are exaggerations of sound normally present in certain individuals—indeed in the case of S<sub>3</sub> present in the majority of persons by graphic method when appropriate low frequencies recording systems are used. The difference is a quantitative one, the normal fades imperceptibly into the pathologic. Often the line can be drawn only with difficulty. Clinical observation supplemented with helpful but not conclusive tests is likely to remain the only means for drawing the line. In adults it is easier to interpret a gallop as such because of the rarity of normal third and fourth sound. Furthermore a presystolic gallop at any age can be interpreted as such since normal atrial heart sound are not usually readily audible at any age."

The protodiastolic gallop usually occurs in conditions of diastolic overload of the ventricle either relative or absolute. For example a loud third sound gallop is characteristic of mitral regurgitation (Fig. 122 and 133) a condition in which the volume of blood entering the ventricle during diastole is increased—the volume represents something approaching a normal quota of blood plus that amount which was regurgitated during the previous ventricular systole. In this instance the ventricular overload is absolute. In myocardial disease which excludes of mitral

regurgitation the condition in which the protodiastolic gallop is most likely to occur the diastolic overload is relative—the normal stroke volume represents an overload for the diseased ventricle.

Protodiastolic gallop. It is usually loudest at the apex or left fifth intercostal space but may be well heard at the femoral border. They are usually of low frequency composition. Often the two are usually felt as heard. The precordium and back may have an extra impulse synchronous with the gallop sound. It is usually a good principle in listening for gallops not to try to hear an extra sound but rather to listen for the general cancer rhythm that characterizes gallop. Unusually in Western cowboy cinema and television programs the rhythm of the gallop is familiar even in the modern urban era.

Tricuspid gallops are likely to be louder in the vicinity of the sternum. They may be loud even in the aortic area. Again they are of low frequency content although in general perhaps more rhythmic than are protodiastolic gallops.

The presystolic gallop seems to occur mainly with condition of systolic overload of the ventricle as systemic arterial hypertension (Fig. 134) aortic stenosis (Fig. 135) pulmonary arterial hypertension pulmonary stenosis (Fig. 136). Thus an occasion is the basis for the fact that the presystolic gallop is termed *bruit de brightie* (from its occurrence with Bright's disease) by French authors of the last century and *kidney gallop* (kidney gallop) by the Germans during the

"Lian (11) also speaks of galop pré-systolique recorded by phonocardiograph which is similar in frequency to the recorded vibrations preceding the summit of the R wave of the electrocardiogram in some patients and not in normal persons. Leonard and co-workers (8) find that the ordinary presystolic gallop can be caused to move toward and into the first part of the first heart sound; the application of force imparts to the extremities. The observations indicate that the initial vibrations of the heart at times are indeed of atrial origin although their persistence in atrial fibrillation suggests that in some cases at least they have other basis. Dunbar (13) concluded that the interval between the presystolic gallop and the first sound is an indication of the severity of the cardiovascular decompensation. When a patient with a presystolic gallop improved the gallop moved progressively into the first sound."

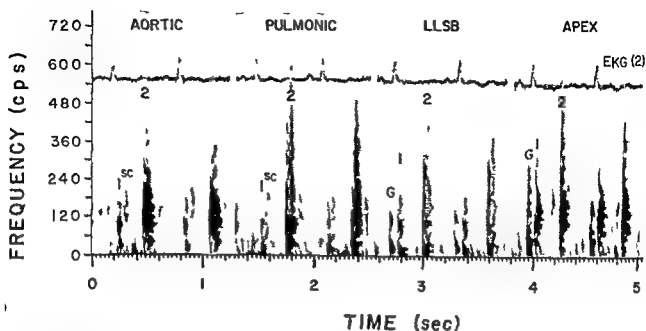


FIG 134 Presystolic gallop

Recorded at indicated areas in W. B. (693724) 40 year old patient with malignant hypertension. At the base there is a presystolic click (SC) which is undoubtedly of aortic origin. At LLSB and the apex the presystolic gallop (G) is striking.

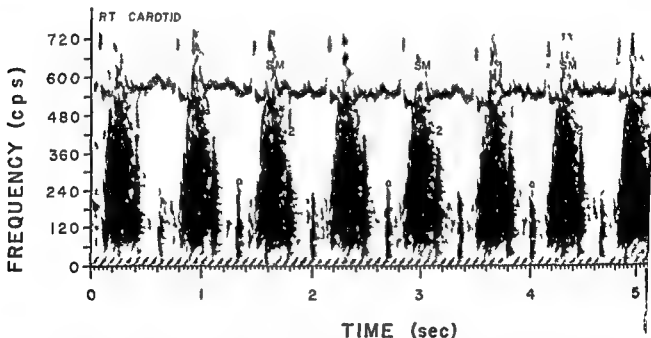


FIG 135 Calcific aortic stenosis with atrial gallop recorded in the neck

1 A. M. D. (411599) 73 year old white female has calcific aortic valve disease. B.I. is 206/76. There was no definite aortic diastolic murmur. The pulses were of water hammer type and there were capillary pulses. She was anemic with hematocrit of 30 per cent. This and arterial sclerosis may account for the wide pulse pressure.

The right carotid record shows the transmission to the neck of the not very murmur and an atrial sound. Precordial records showed the musical murmur characteristic of the Gallavardin phenomenon (p. 267).

That the atrial gallop is right-sided in origin because it was audible almost exclusively in the neck could not be concluded with certainty. Possibly some left atrial gallops are also more likely to be audible in the neck than at the precordium (See fig. 135B).

## THE TRANSIENTS

same period. Perhaps surprisingly, Weitzman (1973) failed to find atrial gallop with increased frequency in aortic valve disease. The remainder of his experience is consistent with mine. He found it frequently in hypertension but only when ventricular hypertrophy was present. However, of those patients with the 4th 77 per cent were free of congestive failure and 23 per cent were totally asymptomatic. When the PR interval is prolonged an atrial gallop is especially likely to occur (a23) or at least is more readily audible (see below).

Because the second sound is often accentuated in the case in which pre-systolic gallop occurs and because the pre-systolic gallop is likely to be taking at the base the rhythm of the pre-systolic gallop is often anapestic (1191) in terms of meter: short short long ( ) ( ) ( ), the accent being on the second heart sound. Some have considered it a helpful mnemonic trick to refer to the pre-systolic gallop as a Tennessee gallop and to the protodiastolic gallop as a Kentucky gallop, the accent being on the second heart sound in each case. It is only fair to point out, however, that the device is often more confusing than helpful. In some areas of the precordium either type of gallop may resemble either Tennessee or Kentucky. This device is possible as a device as the proposal made in 1881 by Braunfeld (477) to classify gallops as hunting, ordinary, or school depending on the type of gait most resembled (709). Although one would scarcely recommend it seriously as a mnemonic device. More code for *m* (—) and for *f* (—) would be as valid as some of the tricks suggested.

Because of the difference in the clinical setting in which the two types of gallop occur the prognostic significance is different. Excluding the protodiastolic gallop of mitral regurgitation protodiastolic gallops have more grave prognostic connotations than do pre-systolic gallop. On an average in adults the life expectancy with a protodiastolic gallop is three to four years (604) which although longer than previously thought is a grave prognosis.

With carotid sinus pressure (588) one may because of the slowing of heart rate and pro-

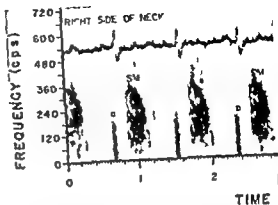


Fig 13B A very similar finding (see fig 13A) in L.C. (1930/122) age 71 who has little evidence of obstruction at the aortic valve but has an exceedingly loud mitral aortic systolic murmur. At the base of the neck there is a pre-systolic gallop.

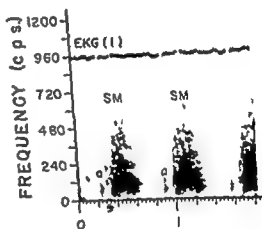


Fig 13A Pre-systolic gallop ( ) in a patient with pure pulmonary stenosis. Note Christmas tree configuration of the systolic murmur and the absence of a gap between the end of the murmur and the aortic component of the second sound (which is buried in the end of the murmur).

longation of diastole be able to determine whether a gallop which before could only be called *m* or *d* is pre-systolic or protodiastolic, or if a quadruple rhythm becomes evident one can conclude that the gallop was of the summation type. With carotid sinus pressure a protodiastolic gallop may disappear through improvement of myocardial competence at the slower rate (1250). Laubry and Harvier (849) used the bradycardic effect of ocular pressure in the study of gallops

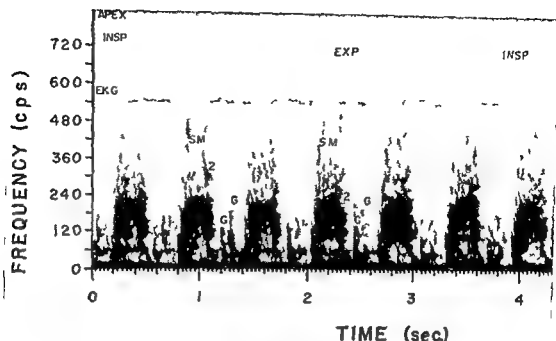


FIG. 137 Double gallop

Both protodiastolic and presystolic gallops in T S (670361) 35 year old female with hypertension posterior myocardial infarction probable multiple pulmonary emboli and congestive heart failure. The PR interval measured 0.23 sec. Note that the systolic murmur has a tendency to murmurality.

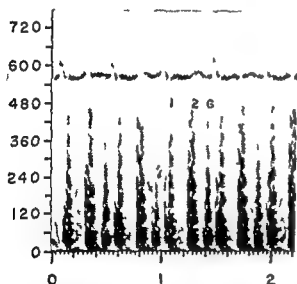


FIG. 138 Mesodiastolic gallop probably summation type. The patient (C C 196903) had severe systemic arterial hypertension.

Carlgren (23a) studied gallops with the multiple filter system of Münchmeyer (calibrated phonocardiography) and concluded that gallops have a predominant frequency in excess of 100 cps as compared with the normal counterparts which tend to lie below this level of frequency. Although possibly statistically true the distinction is not great enough to permit differentiation of

gallops and normal sounds in the individual case. Furthermore, Frost (197-498) could not corroborate Carlgren's findings.

Doek (358) has described a presystolic sound gallop if you will over the jugular veins associated with high venous pressure. This sound appears to be caused by a centrifugal wave from the atrium when there is sinus rhythm and either tricuspid stenosis or elevated end-diastolic pressure in the right ventricle. Often a presystolic gallop was not demonstrable over the precordium. Usually a faint *a* wave was present in the neck veins with the sound near its peak. The sound occurred 0.10 to 0.16 sec after the onset of the P wave of the EKG. Our experience would not indicate that the sound recorded by Doek is specific to the conditions he mentioned or of particular diagnostic utility. Groedel and Miller (604) found the sound frequently and in many different conditions. We have found it for example in calcific aortic stenosis, possibly merely because auscultation in the neck is more regularly practiced in these patients who in addition are prone to have a presystolic gallop (see Fig. 135A and B).

The atrial wave of the apex cardiogram is of large size in cases of presystolic gallop (128-876)

This may support the view that the pre-systolic gallop is a vibration generated in the ventricle as a result of atrial systole.

Duchosal (377) enumerated two laws with reference to the PC interval (the interval between the I wave of the EKG and the atrial gallop).

1 The duration of the PC interval is an index of the seriousness of the affection.

2 The PC interval varies in proportion to the amelioration or the aggravation of the case during its evolution.

Duchosal stated further: We are of the opinion that the variations in the IC interval arise from the variations in the force and volume of the ventricular wave, while the hypotension regulates the production and the acoustic quality of the sound. Muscular hypotension of the ventricle is necessary for the production of gallop rhythm.

Leonard and colleagues (876) have provided convincing confirmation for Duchosal's laws by demonstrating that the PC interval can be reduced by impounding blood in the extremities with tourniquet.

Duchosal (376) points out that the audibility of a pre-systolic gallop is dependent on at least four factors: (1) The duration of the gallop sound, (2) the PP interval, (3) the IC interval, and (4) the R-S<sub>1</sub> (Q-T) interval. Leonard *et al* (875) demonstrate that S<sub>1</sub> is often delayed in hypertension and suggest that increased audibility of a pre-systolic gallop in hypertension can be attributed in part to this fact.

At various times a biventricular gallop that is a gallop sound produced in each ventricle which in the composite may resemble a short murmur has been suggested (336). Although a distinct possibility, such a double gallop awaits more detailed study for confirmation.

The gallop is both one of the most important and one of the most difficult of the auscultatory phenomena. Some clinicians consider it adequate basis for diagnosis; they even question an impression of heart failure if a gallop is not present. It is not present after light exercise. Some would not diagnose gallop if the rate is not in excess of 90, certainly 100. Others would consider it a gallop any exaggeration of a normal diastolic sound. Such exaggerations can occur at

slow rates and are in fact more significant under the circumstance.

THE EARLY DIASTOLIC SOUND IN PERICARDIAL DISEASE. In constrictive pericarditis a sound in early diastole (see pp 127 and 419) may be very loud, even louder than either the first or the second sound. Probably this sound is of the same nature as the protodiastolic gallop. Its character, such as the close position to the second sound, is probably determined by the physiologic conditions peculiar to this disease, e.g., the high venous pressure and early arrest in diastolic filling. In pericardial effusion there occurs a protodiastolic gallop indistinguishable from that of myocardial disease (1078) or with timing and other characteristics intermediate between those of ordinary protodiastolic gallop and the sound of constrictive pericarditis.

The protodiastolic sound of constrictive pericarditis is more likely to occur when the pericardial scar is calcified. It may, in combination with the second sound, simulate a split second sound.

### SYSTOLIC CLICKS

Clicks may be early systolic, mid-systolic or late systolic. They may be single or multiple. They can be further classified on the basis of their seeming mechanism of genesis (see p 129).

1 Clicks caused by pleuropericardial adhesion (usually late, occasionally mid, rarely (186) protosystolic) (See Figs 139 to 144).

2 Clicks caused by costochondral or chondro-sternal motion (usually mid-systolic) (See Figs 145 to 149).

3 Clicks caused by dilatation of hypertension and other diseases of (a) aorta, (b) pulmonary artery (always early systolic) (See Figs 151 to 161).

4 Miscellaneous: (a) Atrial sound in systole caused by low nodal pacemaker with retrograde conduction (ectopic atrial gallop) (176, 686), complete heart block (338), atrial flutter (Fig 443), (b) mediastinal emphysema, (c) left pneumothorax, (d) plaques of calcification in the pericardium (see Fig 429), (e) caused by impact of expanding ventricular aneurysm (Fig 156) or ventricular appendage (Fig 155) on pericardial structure (early or mid systolic).

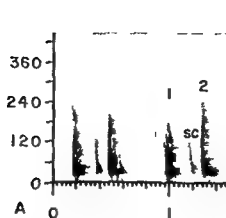


FIG 139

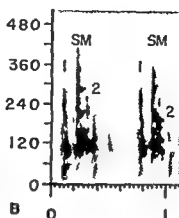


FIG 140

FIG 139 Spectrogram of midsystolic click presumably caused by pericardial adhesion. FIG 140 Same introduction during systolic murmur which is thought to be extracardiac in origin probably caused by pericardial roughening.



FIG 141A Midsystolic click with fibrothorax

N. S. (153008) 53 year old male had pulmonary tuberculosis resulting in left sided fibrothorax which produced an immobilized retracted left hemithorax and drew the heart to the left. The S1 CG shows a midsystolic click (see Fig. 141B)

Among all cases of gallop rhythm studied by Thompson and Levine (1472), 16 per cent had an extra sound in systole responsible for the extra. Although this provides some notion of the relative frequency of systolic clicks it must be noted that early systolic clicks and probably some late systolic clicks would not be counted in a listing of cases of gallop rhythm.

The quality of systolic clicks was well described by Potvin (1224), who referred to them as "small, sharp, clicking sounds, well localized and such that one can scarcely attribute them to anything except the tensing of a pericardial adhesion."

In the spectral phonocardiogram (see the many accompanying illustrations) systolic clicks are characterized by (1) short duration (2) relatively pure frequency content and (3) the fact that the "frequency bottom" usually is not at zero. These spectrographic characteristics are shared with the mitral and tricuspid opening snap (p 191 ff) and with the pulmonary reversal snap of pulmonary stenosis (p 374). Do the valve closure sounds truly have components down to the frequency of a few cycles per second? How many cycles or what fraction of a cycle is necessary to activate the display mechanism of the spectrograph? If one complete cycle is necessary for example, then a 20 cps vibration would need to be at least 0.01 sec in duration to be represented. Clicks and snaps may be too brief for their low frequency components to be demonstrated. On the other hand because the valve closure sounds are commonly palpable it is plausible that they should be found to have subaudible frequency components. It is likewise plausible that snaps should have relatively pure frequency composition well up in the audible range. In brief it is likely that snaps have little or no subaudible frequency composition however even if subaudible components of very low intensity were present, the brief duration of

## THE TRANSLATS

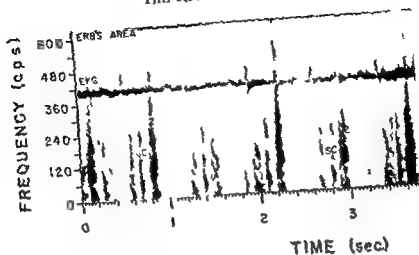


FIG. 14B (See Fig. 14A)

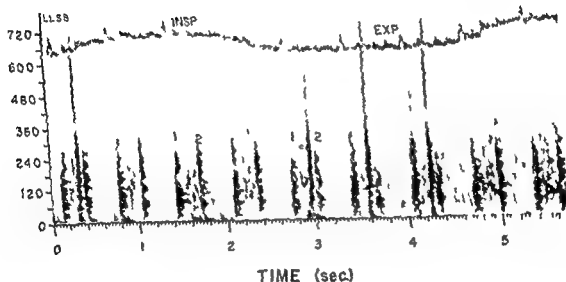


FIG. 14 Late systolic click only in expiration L1SB W. not clear M W (71037) had new onset of endocarditis of the aortic valve

the sound would preclude demonstration in the spectrogram as now made.

The click due to pleuropericardial adhesions are usually loudest at the apex or the lower left lateral border. This type of click constitute the bruit de triquet of Charcot and is the *claquement méso-stérique pleuro-pericardique* of Linn (1920). These clicks may with the second sound produce a combination which can be confused for a widely split second heart sound or a second heart sound plus mitral opening snap. The latter bit of mimicry may of course suggest mitral stenosis

(917). Occasionally as one would anticipate patients with well confirmed mitral stenosis (e.g. M W 726135) have a late systolic click in addition to the signs of mitral stenosis. Clicks of this variety are frequently encountered as a more or less permanent indication of acute pericarditis of rheumatic (108) or idiopathic origin. With respiration or position of the patient there may be variation both in the intensity and timing (70) of the click (Figs. 142 and 143). Sometimes a short murmur is initiated by a click of this type and sometimes the murmur is mitral (p. 207).



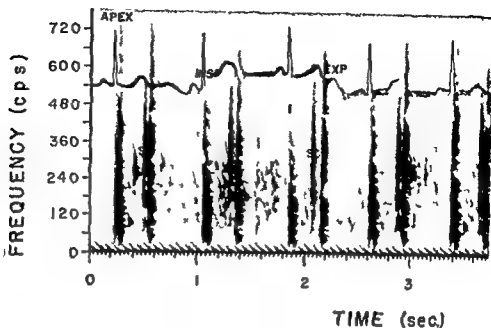


FIG. 143 Late systolic clicks varying in timing with respiration

Incidental finding in T R (171366) 30 year old female with a normal heart. This recording at the apex shows that with inspiration the click occurs earlier in systole.

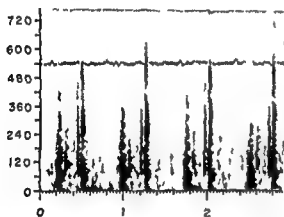


FIG. 144 Early and late systolic clicks

Recorded in a 55 year old woman (699712) who had ARI in youth and displayed mild trichterbrust.

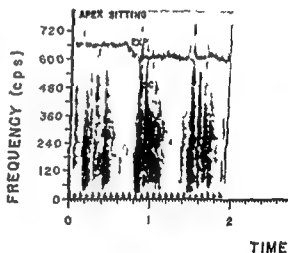


FIG. 145 Multiple clicks with the Marfan syndrome

D D (755872) age 16 years has slight dislocation of the lens and a habitus compatible with the Marfan syndrome. Recording at IISB with patient sitting. At the apex although the main clicks were in systole as they were at IISB there were also less conspicuous ones in diastole.

This type of murmur which is introduced by a systolic click whether noisy or musical is thought to be produced by rubbing of pericardial surfaces. Clicks of probable pleuropéricardial origin may occur in early diastole in patients who also have click(s) in systole (76A).

Systolic clicks due to costochondral or chondro-sternal motion are heard in three types of situation, occurring singly or in combination. First they occur in persons with pectus excavatum, pectus carinatum ('pigeon breast') and other types of chest deformity in which even the normal heart is likely to impinge on the thoracic cage.

In the Marfan syndrome (Figs. 145, 146 and 147) such clicks are particularly frequent in part because of the occurrence of these chest deformities, in part because of the loose jointedness which extends to thoracic structures and of course at times because of enlargement of the heart. Secondly they occur in patients with normal chest walls and normal hearts but a heart

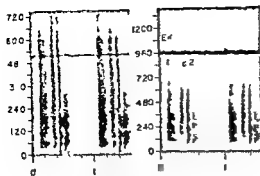


Fig 14b Multiple systolic clicks with the Marfan syndrome

Apex in MI III (60°/662) 46 year-old woman with Marfan's syndrome aortic regurgitation left ventricular enlargement. The multiple systolic clicks were described by the technician as a systolic crunch. Because of the mild chest deformity, cardiac enlargement and general loose jointedness associated with the connective tissue disorder the systolic clicks are thought to have resulted from movement of joints of the bony thorax.

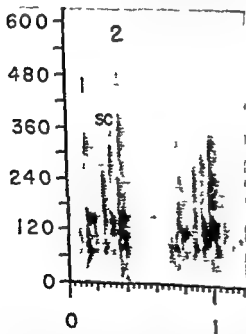


Fig 14c Pulmonary area in patient (MI B 67107) with multiple systolic clicks caused by intra abdominal malignancy heart presumably normal. The multiple systolic clicks probably are caused by movement of costochondral and chondro-sternal joints by the displaced heart.

displaced for some reason such as multiple cysts (Fig 14c) or pregnancy. Thirdly they occur in patient with a normal thoracic cage but an enlarged heart.

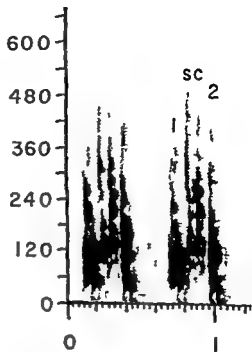


Fig 14d Typical xiphocostal crunch at ILSB in QF (7549). Confusion for a pericardial friction rub occurred when the patient was admitted with fever of uncertain origin.

It is possible that in rare instances a click in early diastole of costochondral and/or chondro-sternal origin (Fig 14d). This category includes the xiphocostal crunch (Fig 14d).

An early systolic click occurs at the base of the heart in a relation with disease of the great vessel. In dilatation and/or atherosclerosis of the ascending aorta especially with systemic arterial hypertension an early systolic click is heard in the aortic area (Fig 150 to 152). In dilatation of the pulmonary artery (Fig 153 and 154) especially with pulmonary hypertension an early systolic click (called pulmonary ejection sound by Leatham and Vogelpoel (86)) is a very frequent and often very conspicuous finding. The proximity of the pulmonary artery to the anterior chest wall may account for this phenomenon's being a more striking feature with disease of the pulmonary artery than with corresponding disease of the aorta. The early systolic click is loudest in expiration. It may give an impression of an unusually early first heart sound since the first sound itself may be inconspicuous at the base.

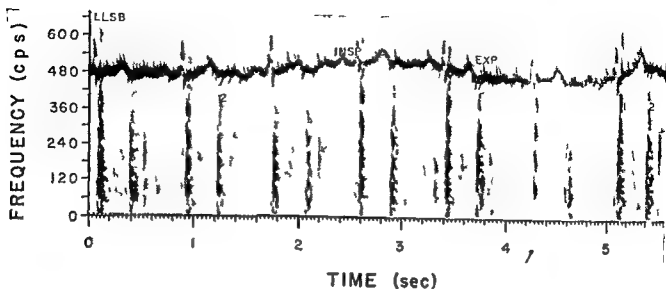


FIG 149 Early diastolic click of extra-cardiac origin

H H (703102) 41 year old female had a typical xiphosternal crunch displayed better in records from other areas. Of particular interest in this recording at LLSB is the early diastolic click. Diastolic clicks of extra-cardiac origin are demonstrated also in Fig 145

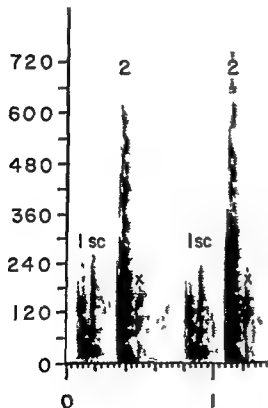


FIG 150 Early systolic click

Aortic area in 33 year old patient (A W 672911) with systemic arterial hypertension with mild dilatation of the aorta and "kinked" carotid. A<sub>2</sub> is accentuated in intensity and frequency span. There is a striking protosystolic snap followed by a short murmur. The sound in early diastole (X) is of uncertain origin. It is probably too close to A<sub>2</sub> to be a protodiastolic gallop. A similar early diastolic sound may be demonstrated in Figure 134

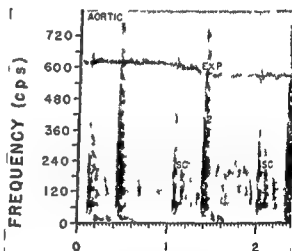


FIG 151 Early systolic click with the Marfan syndrome

W M (702109) 27 year old male has well established Marfan syndrome and is a member of a family with several affected members. A faint aortic diastolic murmur at Erb's area with some enlargement of the left ventricle is present but the aorta is not dilated by x-ray and the patient is asymptomatic. The record here from aortic area shows an early systolic click which becomes most inconspicuous in inspiration. It is probably related to the dilatation of the base of the aorta which is also responsible for the aortic regurgitation

Even in phonocardiograms the early systolic click can masquerade as the first sound. The abnormally great distance between the beginning of the QRS and the extra sound is a clue that it is not S<sub>1</sub> but when there is a question of the presence

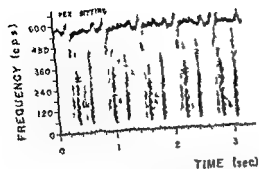


FIG 152 Early systolic click in patient with mild aortic stenosis and aorta bound in false aneurysm after resection of aneurysm caused by cystic medial necrosis (1954) Apex in A II (14034) who 18 months previous to this operation developed

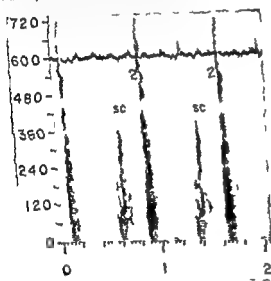


FIG 153 Early systolic click

Pulmonary area in patient with pulmonary hypertension and dilated pulmonary arteries. The early systolic click indicates a faint systolic murmur. An even fainter early diastolic murmur immediately follows the accentuated S<sub>2</sub>.

of mitral stenosis it may be difficult to be certain whether the sound is a delayed mitral closure sound or a pulmonary early systolic click. In the MCC the frequency characteristic (see above) permit differentiation in the majority of instances. When the first sound is pre-systolic followed closely by the systolic click, an impression of split first heart sound is likely to be created. For example others (888 Fig 34) pre-systolic what they interpret as a widely split S<sub>1</sub> in atrial septal

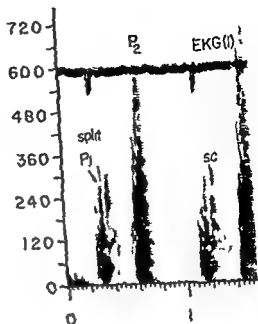


FIG 154 Late systolic click

Pulmonary area in patient (170) (63119) with atrial septal defect and pulmonary hypertension. The accentuated and split the second component (pulmonary closure sound) being the lower S<sub>1</sub> split and followed by a protosystolic click. There is a triple complex in the vicinity of S<sub>1</sub>. This patient subsequently was found to have an atrial septal defect of the ostium primum type with no abnormality of the AV valve.

defect it is rather clearly S<sub>1</sub> followed by an early systolic click.

In addition to dilatation of aorta and hypertension in aortic valve disease increased volume of flow seems essentially to be a feature of cases in which an impressive early systolic click occurs. For example the pulmonary click, particularly striking in cases of atrial septal defect (Fig 154) and left-to-right shunt despite little elevation of pulmonary pressure. Furthermore in tetralogy of Fallot especially with pulmonary stenosis or severe pulmonary stenosis so-called pseudo-truncus arteriosus in which most of the blood is passing out the deformed aorta there is likely to be a click over the aorta heard best at the left of the sternum (975). After Blalock-Tussig or Potts operation the early systolic click tends to be especially pronounced.

Doek (357) and Leitham (862) state that the early systolic click occurs later if diastolic pressure is very high in the pulmonary artery. The interval between the onset of the QRS (or S<sub>1</sub>) and the click

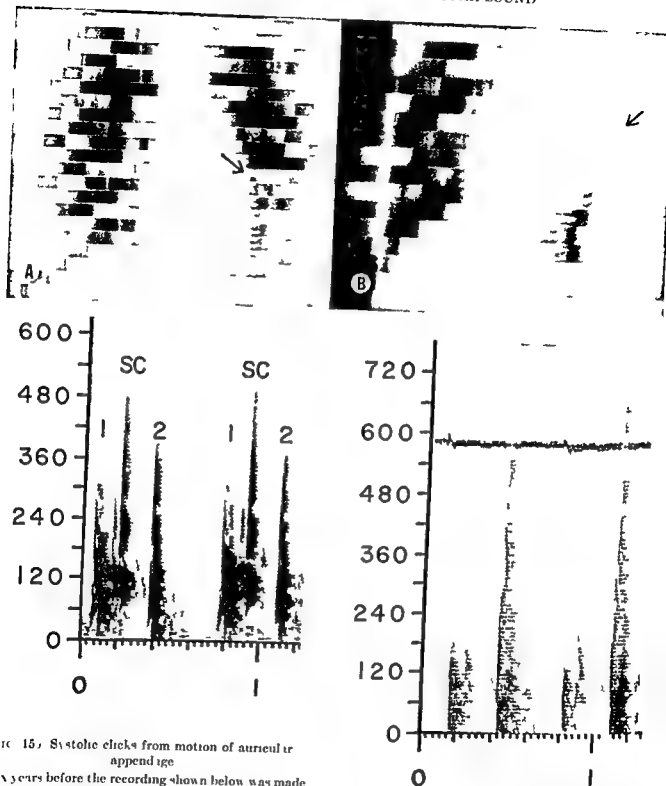


FIG. 15. Systolic clicks from motion of auricular appendage

Six years before the recording shown below was made the patient (I H 452300) had staphylococcal endocarditis on the mitral valve previously damaged it was thought by rheumatic fever. On fluorocopy and roentgen kymography (postero anterior (A) and IAO (B)) the left auricular appendage uncoiled rose out of its bed in the AV groove and probably expanded as did the rest of the atrium. The arrows indicate the areas of left atrial outpouching synchronous with ventricular systole as indicated by inward movement of the ventricular border. The peculiar movement was probably responsible for the peculiar sound.

FIG. 156 Early systolic click with ventricular aneurysm

II SB in W D (654106) 50 year old with old antero lateral myocardial infarction and ventricular aneurysm at lower left heart border. Expansion of the aneurysm in early systole is possibly the mechanism of the sound. In spite of the loud second sound the patient was normotensive. The finding may be the result of valvular sclerosis. Furthermore it is also possible that the early systolic click is of aortic origin and not produced at the ventricular aneurysm.

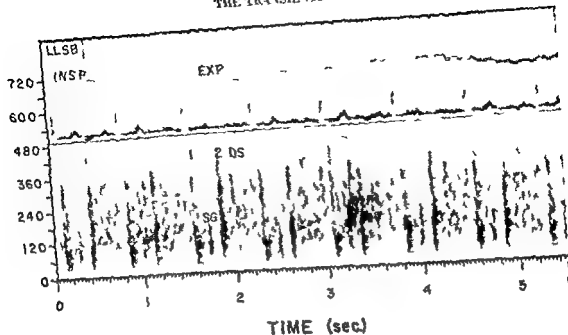


FIG. 15.  $S_2$  tolu gallop in aortic regurgitation

LLSB in O M (04.3) 24 year old patient with a history of acute rheumatic fever at the age of ten years. There were peripheral signs of marked aortic regurgitation. The mid  $S_2$  tolu sound is more than 10 times the usual  $S_2$  tolu click. There is a loud decreased to dia tolu murmur of aortic regurgitation.

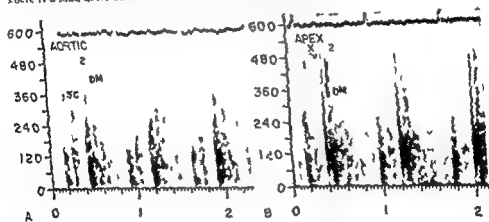


FIG. 16. Circumcribed  $S_2$  tolu sound in syphilitic aortitis with aortic regurgitation

In the aortic area (A) of this patient there is demonstrated a descending murmur in dia tolu beginning immediately with  $S_2$ . Early in  $S_2$  tolu there is a circumcribed sound marked by a  $S_2$  tolu click which is not actually a click as far as either its spectral pattern or the impression it made on the ear are concerned. To the ear there appeared to be a splitting of the first sound.

At the apex (B) of the same patient there is a tele  $S_2$  tolu click (X). In the spectrogram this is clearly a click because of its homogeneity of intra its frequency pattern its brevity and the fact that its frequency bottom does not quite reach the base line as the case with the valve closure sounds. To the ear this sound created the impression of a split second sound & parasternal origin is suggested.

as an index of isometric contraction which may be prolonged with pulmonary hypertension. The interval usually of the order of 0.07 sec. In mild or moderately severe cases of pulmonary

stenosis an early  $S_2$  tolu click occurs but is earlier than in cases of pulmonary hypertension and earlier than the early  $S_2$  tolu click of dilated aorta (866).

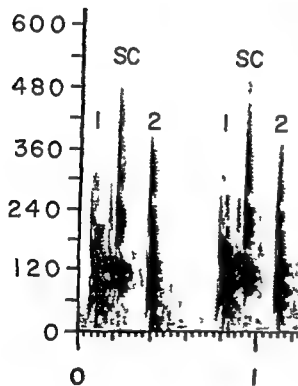
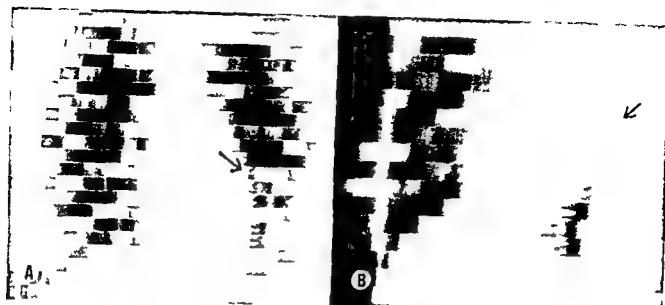


FIG 155 Systolic clicks from motion of auricular appendage

Six years before the recording shown below was made the patient (I H 452300) had staphylococcal endocarditis on the mitral valve previously damaged it was thought by rheumatic fever. On fluoroscopy and roentgen kymography (postero anterior (A) and LAO (B)) the left auricular appendage uncoiled rose out of its bed in the AV groove and probably expanded as did the rest of the atrium. The arrows indicate the areas of left atrial outpouching synchronous with ventricular systole as indicated by inward movement of the ventricular border. The peculiar movement was probably responsible for the peculiar sound.

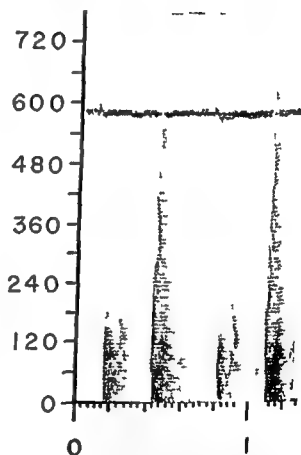


FIG 156 Early systolic click with ventricular aneurysm

156 B W D (681106) 50 year old with old antero-lateral myocardial infarction and ventricular aneurysm at lower left heart border. Expansion of the aneurysm in early systole is possibly the mechanism of the sound. In spite of the loud second sound the patient was normotensive. The finding may be the result of valvular sclerosis. Furthermore it is also possible that the early systolic click is of aortic origin and not produced at the ventricular aneurysm.

consist of at least two closely timed clicks. This mechanism was thought to be impact of the cardiac appendage on the pericardium. In 1957 when the patient was 54 years old he had staphylococcal endocarditis of the mitral valve which was cured with penicillin. While he was still convalescing it was noted on fluoroscopes that there was a bizarre outpouching of the left auricular appendage with each mitral closure click. This has persisted throughout the last 12 years during which time the patient has borne four children and

displayed no symptoms or evidence of cardiac enlargement. The outpouching has been noted and documented by means of roentgen kymogram (Fig. 11) 1 and 2) and cinefluorogram. Clinically the  $S_1$  click is found to correspond precisely with the inflection of the auricular apex click. The  $S_2$  click is louder in the left precordium somewhat removed from the apex click which may account for the lack of much  $S_2$  click murmur of mitral regurgitation in the record of Figure 11, C.

There is a question whether there is anything legitimately termed a mitral gallop.  $S_1$  click may result in a combination with a  $S_2$  or  $S_3$  rhythm when tachycardia is for some reason present. I have avoided the term  $S_1$  mitral gallop because of the benign nature of what we in lead term  $S_1$  clicks and the lack of the relatively grave prognosis associated with the mitral gallop. Most  $S_1$  clicks are just that and by their clicking nature usually differ in quality from gallop sound. Wolferth (1971) believes that aside from the true clicks there are occasional more "blurring" sound in  $S_1$  to which the designation of gallop can be ascribed with validity. In 1940 with Morgagni (1970) he described the mid

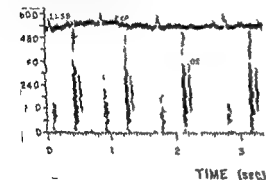


Fig. 100B (For comparison with Fig. 100A) - a typical mitral opening snap of mitral stenosis. From the left lower sternal border in W. S. G. (1955).

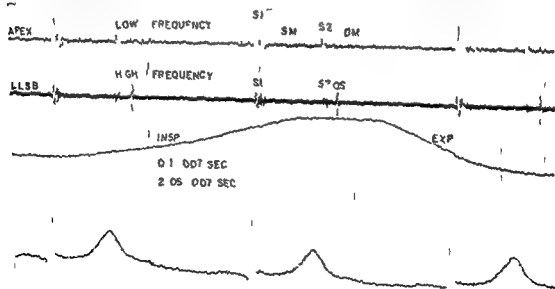


Fig. 161 Opening snap of mitral stenosis

Apex and LLSB in F. T. (1971). 57 year old female. High frequency recordings display opening snaps especially well whereas low frequency recordings are better for demonstrating the diastolic rumble of mitral stenosis. It was for this reason that the recordings from the areas most likely to show one or the other of these phenomena were made with the filter system indicated. The Q1 and Q2 measurements (see p. 152) are indicated.



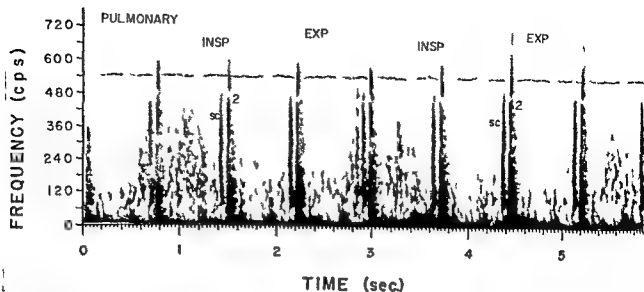


FIG. 159 The late systolic click demonstrated here was loud over the entire precordium in M.C. (11537), 15 year old female who was observed during an attack of acute rheumatic fever over a month previously. During the peak of the attack a pericardial friction rub was present.

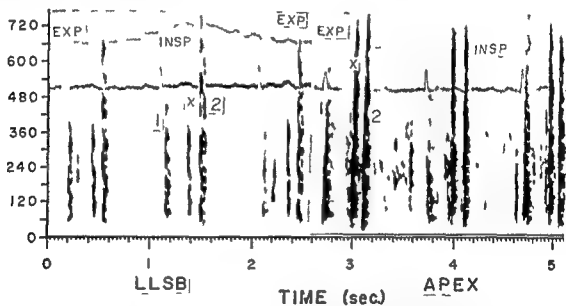


FIG. 160 Late systolic click simulating mitral stenosis.

LLSB and apex in D.P. (B.C.H. 211564) 13 year old female (Photographic record). Mitral stenosis was suspected because of what was stethoscopically interpreted as  $S_2$  followed by an opening snap. The late systolic click is labelled 'X'. The display of the recording from the apex was made with greater amplification so that background noise is more evident and the sound in general appears louder.

Rarely, systolic clicks may be associated with post-infarctional aneurysm of the ventricle (Fig. 156). Although it is possible that pericardial adhesion is the mechanism, it is also conceivable that the impact of the expanding aneurysm on surrounding structures is responsible. The click described by Leroy and Roberts (882) and by Frost (498) was mid-systolic in timing. I have observed an early systolic click in a patient

with an aneurysm origin in the aorta in this atherosclerotic individual cannot be excluded. Manders (1030) describes a similar case.

I have observed a case unique in my experience and possibly in the literature in which mitral regurgitation produced systolic expansion of the left atrium, with uncoupling of the auricular appendage and was associated with a systolic click which graphically (Fig. 155) was seen to

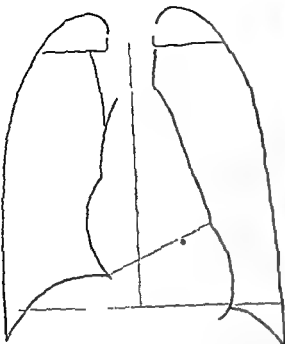


FIG 161 The area of maximum audibility of the opening snap in the chest. The large dot indicates the position in the patient from whom the orthodiagram shown here was made. Difference in heart size and shape were not considered. The lower example was in a patient with a very large heart. Compare the distribution of the dots with the location of the mitral ring as indicated by calcification of the annulus fibrosus mitralis (Fig 319) (Courtesy of Margolies and Wolfert (1940) and *American Heart Journal*.)

opening snap (Fig 160). Finally, the identification of a systolic click in certain areas of the precordium or with certain positions of the patient may suggest the extracardiac and therefore innocent nature of a systolic murmur.

#### ATRIOVENTRICULAR OPENING SNAPS

The last studied is the mitral opening snap of mitral stenosis (Fig 161). Relatively recently (816) the tricuspid opening snap has been characterized.

In mitral stenosis the opening snap occurs shortly after the second heart sound. The length of the interval between it and the opening snap is determined mainly by the level of left atrial pressure or better by the differential pressure or pressure gradient between left atrium and left ventricle at the time of closure of the aortic valve (Fig 162) (Wells (127) thinks the degree of fibrosis in the valve also influences the interval



FIG 162 Predominant mitral stenosis with dense calcification (arrows) in mitral valve. Compare with Margolies and Wolfert's map of the opening snap (Fig 161) (Courtesy of Cooley and Sloan (1911)).

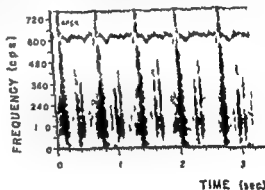


FIG 163 Opening snap as first clue of mitral stenosis.

II S (1932) 60-year-old male has had severe bronchial asthma all his life and shows signs of advanced pulmonary emphysema. An earlier record showed an opening snap as the first suggestion of mitral stenosis. At that time he had a respiratory infection with right-sided heart failure. With clearing of his infection, heart failure and bronchopneumonia cleared. The clinical auscultatory signs of mitral stenosis appeared (see above). The signs were atypical only in that the presystolic murmur was heard amazingly low—at the left costal margin just inside the mid axillary line. Late in systole just before S<sub>2</sub> there is an extracardiac click

(see p 202). Systolic hypertension is accompanied by a longer S<sub>2</sub>-OS interval than would be the case with the same degree of mitral stenosis and no hypertension. The same effect is produced

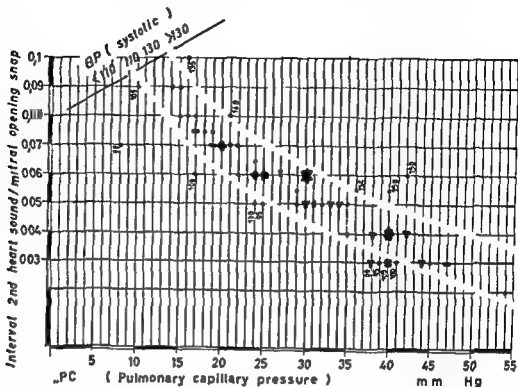


FIG. 162 Relationship between  $S_2$  OS interval and pulmonary capillary pressure in mitral stenosis.

The figures indicate systolic arterial pressure determined by means of the arm cuff. There is a close dependence between the two parameters. With elevated systemic pressure  $S_2$  OS is relatively long with low systemic pressure it is relatively short (from Wolferth *et al.* (1955)).

systolic sound of aortic regurgitation, referring to it is a "systolic gallop." This usage seems entirely legitimate because consistent with its more "thudding" quality, and the SPCG (Figs 157 and 158) shows that the sound extends to the frequency base line as do gallops and valve closure sounds. Its location in mid-systole makes it unlikely that the mechanism is the same as that of the early systolic click of aortic or pulmonary artery origin. The explanation of Wolferth and Margolis (1979)—that it is due either to sudden checking of aortic distension or to impact of the aorta on surrounding structures—seems entirely plausible. Johnston (1961) concluded that whereas pleuropulmonary mid-systolic clicks occur most often at the apex the thudding mid-systolic sound to which the term systolic gallop is applicable occurs most often in the aortic area. Gruehl (1958) also describes a distinctive mid-systolic gallop of aortic regurgitation.

The systolic click is usually of benign significance—the early systolic click is an exception. The main practical clinical reason for familiarity with them is that grave conditions may be simulated. The systolic click, especially with tachy-

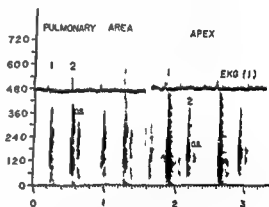


FIG. 163 Mitral stenosis.

Recorded at areas indicated in case with all hallmarks of mitral stenosis except unequivocal rumble. No broad notched P wave of EKG opening up louder pulmonary area apical first sound (1) accentuated both intensity and peak frequency and delayed in relation to QRS.

cardia may create a canter suggesting diastolic gallop. The two may be very difficult to distinguish, with the precise timing of the extra sound determined only by phonocardiography. The late systolic click may with the second sound produce a combination suggesting second sound in

by aortic stenosis and possibly by aortic regurgitation. The interval is shortened immediately after exercise because of rise in left atrial pressure and is prolonged with administration of norepinephrine because of the elevation of systolic pressure in the left ventricle (64). The S<sub>1</sub>-A<sub>2</sub> interval is likely to result in prolongation of the S<sub>1</sub>-O<sub>2</sub> interval. The S<sub>1</sub>-O<sub>2</sub> interval is variable in atrial fibrillation being longer after longer preceding diastolic period (Fig. 163).

The mitral opening snap is best heard in the left midprecordium but may be quite distinct in the aortic area and rather as a rule is louder at the left sternal border than at the apex (Fig. 163). A rather dissipation of the point of maximum audibility on a drawing of the rib cage (1040) tend to show (Fig. 164) a concentration in the area where calcification in the mitral valve is visualized on postero-anterior chest x-ray (Fig. 165).

In combination with the second sound the opening snap usually is a widely split second heart sound. The fact that the opening snap is often loud at the base contribute to this conclusion. This conclusion is probably the basis for the mistaken impression that a split second sound is characteristic of pulmonary hypertension in general and of mitral stenosis in particular. The mitral opening snap usually has a shorter-lived sharper quality than do aortic closure sound however by no means is it always easy to differentiate it by ear from a split second sound. The pattern phonocardiographic feature is its identification.

Fig. 914 emphasizes that auscultation in the upper sternal notch is the differentiation between a split second sound and a second sound followed by opening snap. A mitral opening snap usually well heard in the area. A split second sound usually is not what second sound is heard in the upper sternal notch largely of aortic valve origin.

In the S<sub>1</sub>-C<sub>2</sub> the opening snap has the same characteristics as systolic click (see p. 180). (1) short duration (2) tendency to pure frequency content (3) failure of the 'frequency bottom' to reach zero. The differentiation from the second part of a split second sound is clinched by the demonstration of two separate distinct components of the second sound preceding the open-

ing snap. This is of course most likely to be possible with inspiration and in recording from the pulmonary area where, fortunate for this observation the opening snap is usually well heard and demonstrated.

A prominent mitral opening snap indicates pre-dominant mitral stenosis. As a rule the mitral opening snap is inconspicuous or absent if regurgitation is the predominant mitral lesion. Occasionally a mitral opening snap is present without demonstrable or audible diastolic murmur. However obstruction of a grade to warrant valvulotomy is probably rarely present in such cases. There are exceptions. Recently I inspected mitral stenosis in a man with severe bronchitis. I think because of the presence of an opening snap (Fig. 166). When his asthma improved and presumably his pulmonary vascular resistance was reduced permitting increased flow across the mitral valve a typical diastolic rumble of mitral stenosis appeared (Fig. 166).

The mitral opening snap usually persists after mitral valvulotomy. When valvulotomy has succeeded in relieving the obstruction at the valve the interval between the second sound and the opening snap is increased.

It is doubtful that a normal A<sub>2</sub> opening sound can be heard or recorded at the surface of the chest. However it may be that such a sound will be demonstrated with regularity by intracardiac phonocardiography (p. 84) especially on the left side of the heart. In a variety of conditions in which stenosis and even fibrosis of the A<sub>2</sub> valve is absent a sound suggestive of an A<sub>2</sub> opening snap has at times been heard. Latham (863) comments on it in atrial septal defect. Latham (1975 p. 76) mentions it in patent ductus arteriosus. Latham and colleague (414) in a study of primary pulmonary hypertension. I also have found a sound suggestive of an opening snap in a case of primary pulmonary hypertension with tricuspid regurgitation and have now seen it in a few cases of high flow across the mitral or tricuspid valve in patent ductus arteriosus, atrial septal defect, or ventricular septal defect.

In the S<sub>1</sub>-C<sub>2</sub> a mitral opening snap is often demonstrable in case of overwhelmingly predominant mitral regurgitation but usually such opening snaps situated between the second sound and the third sound gallop are not evident on auscultation.

**TABLL 9**  
**Extra Cardiac Sounds**

|  | Area Heard Best   | Subject Heard In  | Temporal Relationships              | Physiologic Influences   | Mechanism   | Comments   |
|--|---|---|-------------------------------------|--|---|--|
| Split S <sub>1</sub>                           | Apex, LLSB  | Bundle branch block ventricular extrasystoles                                   | Usually 0.05 sec separation         |  | Asynchronism in closure of AV valves because of asynchronism in triggering of ventricles                |  |
| Split S  | Pulmonary LLSB  | ASD bundle branch block reduced lung compliance mitral regurgitation            | Up to about 0.08 sec separation     | Present only in or increased by inspiration (In left b b b decreased by inspiration) | (1) Discrepancy in stroke volumes of ventricles,<br>(2) asynchronism of ventricular triggering          |  |
| S <sub>2</sub> (normal or gallop)              | Apex ILSB   | Children, myocardial disease mitral regurgitation                               | About 0.14 sec after S <sub>1</sub> | Increased by exercise recumbency in expiration                                       | Vibration of ventricular myocardium from rapid filling  | Distinction of normal S <sub>2</sub> from protodiastolic often difficult in young people   |
| S <sub>3</sub> (atrial sound normal or gallop) | All areas   | Children left ventricular failure AV disease                                    | 0.08-0.14 sec after P wave          |  | Components (1) tensing of atrium (2) movement of blood to ventricle (3) vibrations in wall of ventricle |  |
| Mitral opening snap                            | All areas may be least at apex loudest in left mid precordial | Mitral stenosis   | 0.06-0.09 sec after S <sub>2</sub>  | Delayed and less loud after longer diastolic periods in atrial fibrillation          | Abrupt billowing of tenuous mitral curtain toward ventricle sudden check of mitral opening              | Often mis called split S <sub>2</sub>  |
| Isodiastolic pericardial snap                  | Apex ILSB   | Constrictive pericarditis occasional pericardial effusion with tamponade        | 0.08-0.12 sec after S <sub>2</sub>  |  | Abrupt halt in diastolic filling of the ventricle   | Easily mistaken for split S <sub>1</sub>   |
| Systolic click early                           | Base  | Dilated pulmonary artery or aorta especially with hypertension of cor pulmonale | Up to 0.07 sec after S <sub>1</sub> | Loudest in expiration  | Snapping of arterial wall early in ventricular ejection   | May be confused with split S <sub>1</sub> at base if S <sub>1</sub> faint it base may simulate snapping S <sub>1</sub>                 |
| Systolic click especially mid or late          | All areas least at base                                       | Previous rheumatic fever chest deformity such as pectus excavatum               | Variable                            | Location of click in systole may vary with inspiration                               | Pericardial adhesions grating of costochondral or chondrosternal joints                                 | May be multiple late clicks probably most often due to adhesions may be mistaken for S <sub>2</sub> and opening snap of mitral a. oris |

by aortic tension and possibly by aortic regurgitation. The interval is shortened immediately after exercise because of rise in left atrial pressure and is prolonged with administration of norepinephrine because of the elevation of systolic pressure in the left ventricle (63). The  $S_1$ - $S_2$  interval is likely to be short in prolongation of the  $S_2$ - $S_3$  interval. The  $S_2$ - $S_3$  interval is variable in atrial fibrillation being longest after longer preceding diastolic periods (Fig. 163).

The mitral opening snap is best heard at the left midprecordium but may be quite distinct in the aortic area and rather as a rule is louder at the left sternal border than at the apex (Fig. 164). Scatter diagram of the point of maximum audibility on a drawing of the rib cage (1010) tend to show (Fig. 164) a concentration in the area where calcification in the mitral valve is visualized on roentgenographic technique (Fig. 164).

In combination with the second sound the opening snap suggests a widely split second heart sound. The fact that the opening snap is often loud at the base contributes to this confusion. This confusion is probably the basis for the mistaken impression that a split second sound is characteristic of pulmonary hypertension in general and of mitral stenosis in particular. The mitral opening snap usually has a shorter—indeed a snappier—quality than does the closure sound, however by no means it always fails to differentiate it by ear from a split second sound. The pretrial phonocardiographic features assist the identification.

Linn (914) emphasizes that an cultivation in the upper third notch assists the differentiation between a split second sound and a second sound followed by opening snap. A mitral opening snap is usually well heard in the area. A split second sound usually is not well heard in the upper third notch; largely of aortic valve origin.

In the SLC, the opening snap has the same characteristics as systolic clicks (see p. 180): (1) brief duration; (2) tendency to pure frequency content; (3) failure of the frequency bottom to reach zero. The differentiation from the second part of a split second sound is clinched by the demonstration of two separate distinct components of the second sound preceding the open-

ing snap. This is, of course, most likely to be possible with inspiration and in recordings from the pulmonary area, where, fortunate for this observation the opening snap is usually well heard and demonstrated.

A prominent mitral opening snap indicates predominant mitral stenosis. As a rule the mitral opening snap is more conspicuous or distinct if regurgitation is the predominant mitral lesion. Occasionally a mitral opening snap is present without demonstrable or audible diastolic murmur. However obstruction of a graft to warrant valvulotomy is probably rarely present in such case. There are exceptions. Recently I suspected mitral stenosis in a man with severe bronchial asthma because of the presence of an opening snap (Fig. 166). When his asthma improved and, pre-emptively, his pulmonary vascular resistance was reduced permitting increased flow across the mitral valve a typical diastolic rumble of mitral stenosis appeared (Fig. 166).

The mitral opening snap usually persists after mitral valvulotomy. When valvulotomy has succeeded in relieving the obstruction at the valve the interval between the second sound and the opening snap is increased.

It is doubtful that a normal AV opening sound can be heard or recorded at the surface of the chest. However it may be that such a sound will be demonstrated with regularity by intracardiac phonocardiography (p. 84) especially on the left side of the heart. In a variety of condition in which stenosis and even fibrosis of the AV valves is absent a sound suggesting an AV opening snap has at times been heard. Leitham (843) comments on it in atrial septal defect. Linn (917) p. 176 mentions it in patent ductus arteriosus. Evans and colleagues (411) saw it in a case of primary pulmonary hypertension. I also have found a sound suggesting an opening snap in a case of primary pulmonary hypertension with tricuspid regurgitation and have now seen it in a few cases of high flow across the mitral or tricuspid valve in patent ductus arteriosus, atrial septal defect or ventricular septal defect.

In the SLC a mitral opening snap is often demonstrable in cases of overall dominantly predominant mitral regurgitation but usually such opening snaps situated between the second sound and the third sound gallop are not evident on an auscultation.

## CHAPTER 13

# Murmurs

### NOISY SYSTOLIC MURMURS

Latham (856) proposes that systolic murmurs be divided into two general categories (1) *Ejection systolic murmurs*, which are mid-systolic in timing and associated with flow through the aortic or pulmonary valve, (2) *regurgitant systolic murmurs* which are holosystolic (p 50) and which are produced by mitral or tricuspid regurgitation or by ventricular septal defect

*Ejection systolic murmurs* swell to a peak about mid-systole and invariably finish before the second heart sound, or at least before that portion of the second sound produced by closure of the valve where the murmur originates. This gap is to be expected since forward flow must have ceased before the local back flow responsible for valve closure can occur. A murmur of the ejection type originates at the aortic or pulmonary valve under the following circumstances alone or in combination

- 1 Stenosis of valve or infundibulum
- 2 Valvular damage without obstruction
- 3 Dilatation of the vessel beyond the valve
- 4 Increased flow or rate of ejection

Aortic systolic murmurs are usually best heard in the aortic area. However in children with congenital aortic stenosis, the systolic murmur may be loudest at the left sternal border leading to a misdiagnosis of ventricular septal defect. The murmur tends to be well heard at the base of the neck on the right and a frequently confusing feature at the apex. The presence of a silent interval between the end of the murmur and the second sound is a useful point of differentiation from the holosystolic murmur of mitral regurgitation and ventricular septal defect. When the order of closure of the arterial valves is paradoxical as with severe aortic stenosis or left bundle branch block, the murmur may reach

the earlier pulmonary component but not the aortic

Aortic stenosis results in a classical murmur which was earlier referred to by Latham (808) as an *ejection stenosis murmur*. In the oscillogram (Figs 167 and 168) the murmur is diamond-shaped, that is has its intensity peak in mid-systole. In the spectrogram the murmur has the configuration of a Christmas tree since its frequency peak is likewise in mid-systole.

Arizumi and Iwamoto (26-27) find that the peak of the murmur—they used intensity peak but the same statement should apply to frequency peak—is later in systole the severer the grade of aortic obstruction. In fact many of the murmurs included in the general class of ejection systolic murmurs are virtually decrescendo murmurs, so early does their peak occur after the first heart sound. Because of the frequent association of an early systolic click (see p 179) with murmurs of the ejection type the murmur frequently displays a decrescendo fall away from an early systolic click.

In aortic regurgitation an ejection systolic murmur, which sometimes is even associated with a systolic thrill results from a ventricular stroke output which is increased in both rate and volume and which is occurring across a deformed albeit not stenotic valve. Dilatation of the aorta beyond the valve—in aortic aortitis with aortic regurgitation for example—may contribute. It is proper to speak of the murmurs of aortic regurgitation not merely the murmur referring only to the diastolic one.

*Pulmonary stenosis* (Fig 169) is associated with a systolic murmur of a shape similar to that of aortic stenosis but with a peak situated later in systole. Because of prolonged right ventricular systole and much delayed pulmonary valve clo-

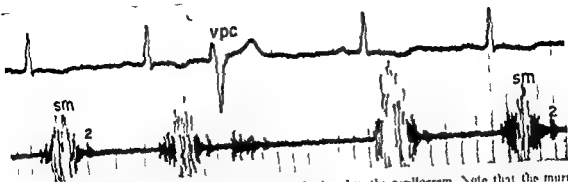


FIG 15—Diamond shaped murmur of aortic stenosis as displayed in the oscillogram. Note that the murmur stops before the second sound that the murmur with the extra systole is much reduced in intensity and that the murmur after the compensatory pause is increased in intensity. (Each of the intervals indicated by the time lines is 0.10 sec.)

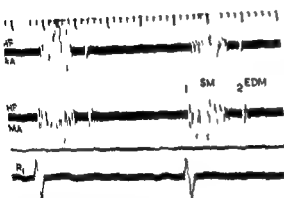


FIG 16a Aortic stenosis

Mild aortic ejection murmur at aortic area (AA) finishing before aortic closure (?). There is an early diastolic murmur (EDM). (Courtesy of Dr. Aulrey Leatham.)

sure the systolic murmur tend to extend into the aortic closure sound and may extend across and drown it. However, since it always stops before the pulmonary valve closure, the murmur rhymes by the pacification laid down for an ejection murmur.

With dilated pulmonary artery an early systolic click is particularly likely to occur and the ejection murmur is likely as stated above to be decrescendo from it. With increased flow in the pulmonary artery—as in atrial septal defect and other example of left-to-right shunt—before the level of the pulmonary valve thyrotoxicosis, exercise, complete heart block with increased stroke volume because of the low rate anemia, exercise in young subjects—the ejection murmur which has its peak soon after the



FIG 16b Pulmonary stenosis

RI pressure 62/5 mm Hg. Mild systolic ejection murmur goes up to aortic closure sound but ceases before late soft pulmonary closure sound.

first sound. Here is another demonstration that the timing of the peak of the murmur is largely dependent on how much obstruction there is to ejection.

A large proportion of innocent (so called functional) murmur heard at the base are probably of the nature of ejection systolic murmurs. They finish before the second sound become louder with increased blood flow of exertion or excitement and tend to disappear as the child becomes older.

The ejection systolic murmur associated with high flow in the pulmonary artery often has a



scratchy quality. In thyrotoxicosis this feature is so striking that the murmur is referred to as a *Lerm in Meins scratch*. It may be confused with a pericardial friction rub. The anatomical super-

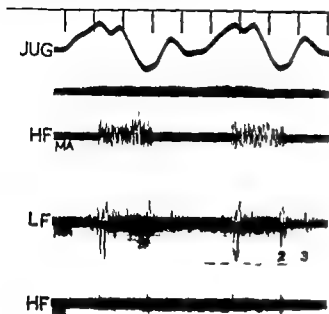


Fig 170 Mitral regurgitation with holosystolic murmur

There is a protodiastolic gallop in the low frequency recording (LF). MA = mitral area, HF = high frequency recording, PA = pulmonary area. (Courtesy of Dr Aubrey Leatham.)

ficiency of the pulmonary artery especially when dilated is probably in part responsible for the superficial quality of this murmur.

Regurgitant systolic murmurs are, with few exceptions (see below), holosystolic, because throughout systole pressure in the donor chamber is higher than that in the recipient chamber. Take, for example the left ventricle and left atrium in mitral regurgitation. There is persistence of a big pressure difference at the time that aortic closure occurs and continuation of the murmur is therefore likely to obscure the second aortic closure sound—at the apex. The exaggerated third heart sound in mitral regurgitation must not be mistaken for the second sound.

As to its shape the murmur of mitral regurgitation (Fig. 170) may be plateau, decrescendo from the first sound or crescendo to the second sound. However it is probably for practical purposes always holosystolic. Viquez (24) thought that functional mitral regurgitation could produce a nonholosystolic murmur. One can conceive of an anatomical situation such that the systolic murmur occurs only when pressure in the ventricle is highest and therefore capable of prying the valve open as it were. This situation is not proved however and possibly is unlikely since

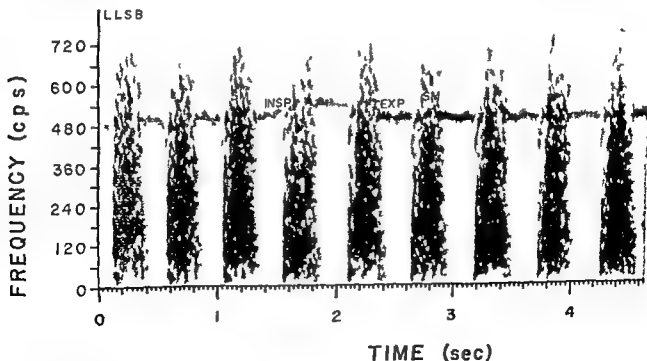


Fig 171 LLSB in C A (757015 B9062) asymptomatic 5 year old male with typical *maladie de Roger* confirmed by cardiac catheterization pulmonary pressure normal

ventricular pressure builds up rapidly in the ventricle and well maintained and fall off rapidly.

The basis for the three different types of systolic murmur is unknown. Particularly it is difficult to understand the hemodynamic and anatomical basis for the configuration of the type of regurgitant murmur which has its peak in late systole in the vicinity of the second heart sound. To the ear these murmurs may seem not to be holosystolic but by graphic means it is possible to demonstrate component of the murmur in early systole. When a murmur is truly limited to the latter part of systole or if it is excruciated (with a brief silent gap between it and both the first and the second sound) if it is introduced by a mid or late systolic click and if it varies appreciably with respiration then differentiation from a regurgitant murmur is provided and extracardiac origin is suspected. The only exception to the statement that circumscribed mid systolic murmurs with a silent interval both before and after the murmur are not always extracardiac seem to be some cases of aortic stenosis. Especially when left bundle branch block is present there seem likely to be a gap between the first sound and the murmur as well as the expected gap between the murmur and the second sound.

The holosystolic murmur of mitral regurgitation is loudest at the apex but radiates well into the left axilla and to the back. With mammoth enlargement of the left atrium unusual transmission of the murmur may occur. It may be heard in strikingly loud form in the back and at times as far as the navel or even the iliac crests and occiput (1478). Conduction in the aortic structure is probably responsible for the unusual left atrium which tends to impinge on the pine and may rarely erode the vertebral bodies like an aortic aneurysm paring the more resistant intervertebral disk. Children and adolescents are most likely to show the phenomenon indicating that an enormous heart in a small chest is probably the responsible factor. When the large left atrium pre-erupts to the right of the sternum with a systolic expirile pulsation and sometimes thrill in that area there may be heard in the same area a systolic murmur which is almost certainly the mitral regurgitant murmur. This creates confusion with other possibilities especially aortic

stenosis and tricuspid regurgitation. The fact that the murmur is loudest at about the level of the third right inter-space and is not transmitted into the neck and the demonstration of mammoth left atrium by radiologic techniques help clarify the diagnosis.

Although characteristically the murmur of an complicated ventricular septal defect (*maladie de Roger*) is holosystolic (Fig 171) it becomes limited more and more to early systole when pulmonary resistance is increased and the left-to-right shunt reduced or eliminated. With a moderate degree of pulmonary hypertension the abbreviation of the murmur gives it the appearance of an ejection murmur. Another complicating feature is that the dilated pulmonary artery and increased pulmonary flow may produce an early systolic click followed by a genuine ejection murmur.

### NOISY DIASTOLIC MURMURS

There are three main varieties of non-musical diastolic murmur: (1) the decrescendo (or diminishing) relatively high pitched diastolic murmur which begins immediately with the second heart sound; (2) the relatively low pitched rumbling diastolic murmur beginning after the second sound by a short interval—the so-called mid-diastolic murmur; and (3) the presystolic or atriosystolic murmur. The first three we will refer to as the *arterial diastolic*, the *passive diastolic*, and the *atriosystolic* murmur, respectively.

*Arterial diastolic murmurs* result from aortic or pulmonary regurgitation. Because of the relatively high pressure in the aorta normally and in the pulmonary artery in condition which are associated with the so-called Graham Steell murmur the arterial diastolic murmur is high pitched, whizzing whurring in quality.

With rare exceptions the arterial diastolic murmur begins immediately with the closure sound of the valve at which the murmur is generated. Holdack and Wolf (706) observed a silent interval in some cases of pulmonary regurgitation and such an interval may occur in aortic regurgitation (see below). Occasionally it increases in intensity and peak frequency a short time before the decrescendo that is, it is decrescendo rather than merely decrescendo (1529). This pattern doubtless corresponds to an acceleration of re-

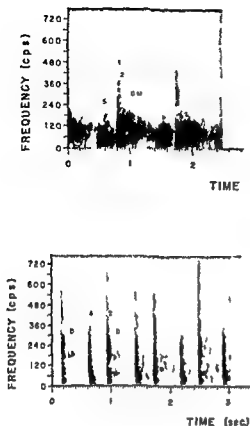


FIG. 172 Typical murmur of aortic regurgitation decrescendo in peak frequency and in intensity (above). Advanced case. The systolic murmur may be caused by aortic regurgitation alone or may indicate a small amount of aortic stenosis (Below). Early case.

gurgitation momentarily before deceleration. It seems that it is regurgitation of mild or moderate degree that it is most likely to result in a murmur of crescendo-decrescendo pattern. The crescendo portion corresponds temporally to the diastolic wave of the aortic pressure pulse (706). A gap heard on auscultation in some cases of aortic regurgitation may have its basis in the crescendo-decrescendo pattern; the former phase may not be appreciated (860). In other cases, paradoxical splitting of the second sound, with the aortic sound after the pulmonary and possibly reduced in intensity, may be the basis. In some other cases the basis is by no means clear. In these instances there appears to be an entirely silent interval between the second sound and abrupt onset of the murmur about 0.10 or 0.12 sec later. Since the mitral valve has opened shortly before with decompression of the left atrium, is it possible that support of the aortic ring provided by the contiguous mitral ring is lost so that aortic regurgitation rather abruptly begins or is increased? The decrescendo diastolic murmur of aortic or pulmonary regurgitation is decrescendo not only in intensity, as is well demonstrated by the oscillogram, but also in peak frequency, as is demonstrated by the spectral phonocardiogram.

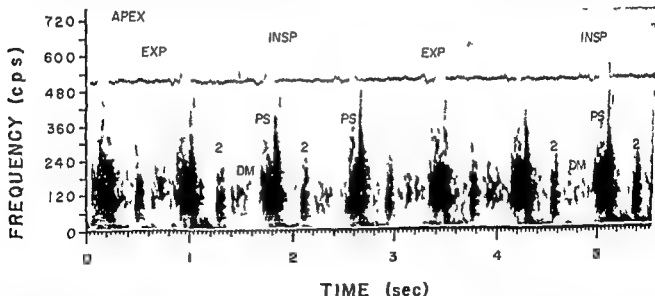


FIG. 173 Mitral stenosis.

Apex in M. K. (H. L. H. 94572), 19-year-old female. There is a minimal systolic murmur. An opening snap is not clearly demonstrated here but was evident in ILSB and pulmonary areas.  $M_1$  is ringing as indicated by the relatively high frequency content demonstrated and is delayed relative to QRS. The two parts of the diastolic murmur—presystolic first part (DM) and presystolic crescendo (PS)—are clearly demonstrated. The presystolic murmur has the appearance of half the Christmas tree, which is characteristic of an ejection stenosis murmur as in aortic stenosis.

The arterial diastolic murmur of aortic origin (Fig. 172) is most often loudest at Erb's point (the third left intercostal space at the sternal border) occasionally at the cardiac apex and sometimes in the left axilla (the Cole-Corral murmur p. 37). Bülthard-Lotter (p. 29) thought that the direction of radiation of the aortic diastolic murmur was an indication of the precise anatomical change at the aortic valve. Particular kinds of echocardiograms predominantly involved. However, recent work of this matter by Herr and Limer (1933) (p. 279) make it seem unlikely that much diagnostic significance should be attached to the direction of radiation of the murmur, although Harvey (1932) was impressed with the predominant radiation down the right sternal border in cases of syphilitic aortitis. The Virchow syndrome and coronary sinus aneurysm in Hoffman and B'out (1930) and White (1930) also wrote that in syphilitic aortitis was down the right sternal border more commonly than in rheumatic aortitis.

The arterial diastolic murmur of pulmonary origin is usually confined to the left sternal margin. From the standpoint of quality there is really nothing to differentiate it from the murmur of aortic regurgitation. Any arterial diastolic murmur heard at the right of the sternum is probably of aortic origin. Furthermore the murmur of pulmonary regurgitation may on rare occasion be influenced by respiration that is it may be accentuated in inspiration. As noted above Holldack and Wolf (1906) observed a gap between  $S_2$  and the murmur in some cases. Usually, however, no such sign such as the peripheral signs of aortic regurgitation must be relied upon to make the differentiation.

The most frequent form of pulmonary regurgitation is that which generates the *functional* murmur. The murmur of high pressure in the pulmonary artery (p. 30) is very rarely does decrease of the pulmonary valve producing pulmonary regurgitation occur in the absence of pulmonary hypertension. When such does occur for example with an isolated congenital defect of the pulmonary valve or damage in gonococcal endocarditis the murmur is likely to be more long pitched and blubbery.

Both type of arterial diastolic murmurs are best heard when the subject is in the sitting position especially if he is leaning forward in full expiration. The examiner may exert effort to placing the patient in the knee-chest position. These murmurs are notorious for being faint and easily missed.

The position of the aortic murmur accompanies stenosis of the mitral (Fig. 173) or tricuspid valve relative or absolute. It is always separated from the second sound by an interval. It may, in the case of organic stenosis, be begun with an opening snap or even in that instance may have its onset somewhat later than the opening snap. The precise diastolic murmur is usually decrescendo in pattern. If the mitral stenosis is sufficiently mild, diastole sufficiently long, or flow sufficiently low, the murmur may actually end before the onset of the aortic diastolic murmur or before the end of diastole in the case of atrial fibrillation. The decrescendo character and the radiation of the murmur are the result of diminishing atrioventricular pressure differential. The simultaneously recorded pressure curves from left atrium and left ventricle obtained by left heart catheterization, sometimes indicate that the reduction of the gradient is more a matter of time in ventricular pressure than of fall in atrial pressure (1939). This would be expected to be the case particularly if there were associated aortic regurgitation or change in the pressure-volume characteristics of the left ventricle resulting from the ventricular hypertrophy of an associated lesion such as aortic stenosis. Elevated left atrial pressure may be maintained by pericardial inflow from the lungs when ventricular pressure will rise unusually rapidly for the reasons outlined.

When the stenosis is of the relative type—that is, the valve is normal but flow across the orifice is increased and the ventricle beyond it enlarged—then the pressure in the left ventricle tend to be introduced by an exaggerated third heart sound. The particular variety will be referred to as the Carey-Coombs murmur because although the eponym was originally assigned to this type of murmur occurring specifically in the course of acute rheumatic fever the murmur of all relative mitral stenosis has the same pattern. In general the pressure in the left ventricle is low pitched and rumbling in quality. The Carey-Coombs murmur often may be better described as blubbery. Flow of blood into the ventricle in

TABLE 10

*A Comparison of the Arterial Diastolic Murmur of Aortic Regurgitation with the Passive Diastolic Murmur of Mitral Stenosis*

|                  | Arterial Diastolic Murmur of Aortic Regurgitation  | Passive Diastolic Murmur of Mitral Stenosis  |
|------------------|--|--|
| Time             | Early immediately following the second sound   | Later beginning, an appreciable interval after the second sound often immediately after the opening snap when it is present it extends through mid diastole if loud if the stenosis is well marked and the rhythm normal it is followed by a presystolic accentuation and thrill especially when the pulse is quickened after exercise |
| Character        | Blowing often high pitched   | Rumbling usually low pitched rarely blowing  |
| Site             | Maximal along the left border of the sternum heard frequently at the apex and rarely maximal at the aortic area (in such a case mitral aortic dilation is usually present) | Maximal at the apex and often limited to a very small area but sometimes heard as far as the sternum   |
| Position of body | Best heard in the upright position leaning forward   | Best heard in the recumbent position   |
| Stethoscope      | Best heard with the diaphragm sometimes it may be heard better with the naked ear  | Best heard with the bell chest piece   |

the first part of diastole seems to be especially likely to produce sound in children. Furthermore a murmur is especially likely to be generated at the mitral valve in hypervolumic states. This may

be the result of higher pressure in the left atrium, which in turn is due to its steeper pressure-volume curve than in the right atrium. However, high flow does generate a passive diastolic murmur at the tricuspid valve in atrial septal defect.

What will be said about the locus of maximum audibility of the presystolic atrio-systolic murmur (see below) applies equally to the passive AV diastolic murmur.

By way of summary, reference can be made to Table 10, which was originally devised by Paul Dudley White in 1926 (1510).

The *atriosystolic murmur* is probably better termed, rather than presystolic, since when there is some degree of atrioventricular dissociation the murmur may not be immediately presystolic (1150). The atrio-systolic murmur is essentially an ejection systolic murmur (see p 194). When the PR is prolonged so that the atrio-systolic murmur stands alone, it is seen that it has the diamond shape in the oscillogram and in the spectrogram has the configuration of the Christmas tree, just as does the much louder murmur of aortic stenosis. When the PR interval is of normal duration the presystolic murmur is half the Christmas tree of an ejection systolic murmur, being cut short by the snapping mitral first sound.

Excluding the Coombs murmur (p 199) which is a different matter the presystolic murmur is often the sole murmur of minimal mitral stenosis. The reason is obvious: it is to be expected that, in mild cases only with atrial systole will there be sufficiently rapid trans mitral flow to produce a murmur.

In the past some (e.g. 904) have questioned whether the presystolic murmur is truly crescendo. One group (13) states as follows: "The acoustic crescendo effect is usually in auditory illusion because of its close proximity to the accentuated first sound. They have been led to this conclusion by failure to demonstrate a clear crescendo of intensity in the oscillographic PCG (p 296). The spectral PCG leaves no doubt that the presystolic murmur of mitral stenosis is indeed crescendo."

The atrio-systolic origin of the presystolic murmur, a matter of debate half a century ago has again been brought in question by one group (1143) which claims that it is in fact a murmur

produced by vibrations of the mitral valve early in ventricular systole as the mitral valve moves toward the atrium. Others have conceived of there being regurgitation at the mitral valve for a brief period before the diaphragm of the stenotic valve snaps toward the atrium with production of the ringing first heart sound. Both theories are discredited if the idea that the murmur is systolic not presystolic. The fallacy of this view, which has no definite evidence to support it in either of its two forms, is revealed by recording of the murmur in mitral stenosis which shows the presystolic murmur beginning well before the QRS of the electrocardiogram. In 1913 Sir Thomas Lewis (904) wrote: "It is generally believed to be presystolic in time, yet from time to time its actual position in the cycle has been hotly contested. I do not propose to pursue a discussion which at the advent of sound record has become almost purely historical. The isolated presystolic murmur with long PR interval is further evidence for the mitral stenotic nature of the presystolic."

In mitral stenosis the two Aortic valve murmurs just discussed—presystolic diastolic and aortic regurgitant—are audible at the cardiac apex often in a confined area; however they are best audible when the subject is in the left lateral decubitus position possibly because the apex is brought into more intimate contact with the anterior chest wall. The exercise of sitting up and lying down several times and then turning on the left side will often bring out these murmurs when they are not otherwise audible. Walking about will sometimes bring out the murmur of mitral stenosis when it is up to the limit. In a patient merely having his cough may be sufficient to bring out a mitral diastolic murmur. Pharmacologic methods, e.g., amyl nitrite and phlebotomy have some time been used to increase flow to the point that the murmur will be heard. The presystolic murmur is likely to be increased by any increase in heart rate—probably because the atrium has less time for decompression before atrial contraction

occurs—and the effects of many measures may be mainly through the effect on heart rate. There may in effect be a summation of the presystolic diastolic and the aortic regurgitant blood flow across the mitral valve may actually fall.

As a rule the diastolic murmurs of mitral stenosis do not radiate widely and are audible only in a very limited area. They usually do not radiate into the left axilla as in the case of the systolic murmur of mitral regurgitation. Occasionally, however, when very loud they may be audible over most of the body of the heart and even in the aortic area. In children and other individuals with small chests this is especially likely to be the case.

The location of the diastolic murmurs of tricuspid stenosis is more variable. They may be heard at the lower left sternal border, in the region of what is considered the cardiac apex or at any point in between. Differentiation from the diastolic murmurs of mitral stenosis is often difficult. Accentuation by inspiration is helpful. Occasionally, however, the diastolic murmurs of mitral stenosis are accentuated by inspiration. The diastolic murmurs of organic tricuspid stenosis—disregarding for the moment the functional tricuspid stenosis of atrial septal defects—tend to be louder than the corresponding murmurs of mitral stenosis are often accompanied by a very striking thrill at the left of the sternum and in the precordium have a greater frequency span than usually even in the murmur of mitral stenosis. The latter does not mean, however, that the murmur sound higher pitched. The features of the murmurs of tricuspid stenosis are probably the result of the more superficial location of the tricuspid generator.

Ristand (133) described what he termed an auricular diastolic murmur with complete heart block in elderly patients. It is preceded by an atrial heart sound.

In mitral stenosis each diastolic murmur at the apex may occur in patients with 2:1 heart block and a low ventricular rate. The murmur has the characteristic of Carey-Coombs murmur. The first uncondensed P wave occurs early in the ventricular diastole. The combination of atrial systole and rapid passive inflow is probably responsible for this murmur. The second P wave

The precise mechanism is unknown. One suggested possibility is that in the left lateral decubitus gravity favors flow through the mitral valve. Harvey (67) has even questioned that there is anything special about the left lateral decubitus and I suggest that the exertion of turning into that position may alone account for the temporary accentuation of the diastolic murmur.

STENOIC L...

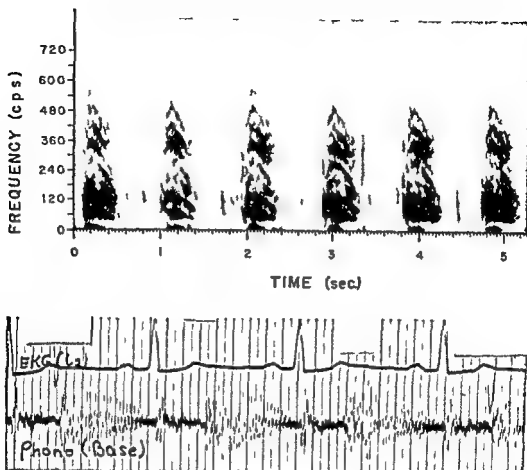


FIG 174 Recordings of a sea gull diastolic murmur of retroerect aortic cusp (Above) Spectrogram (below) Oscillogram made with a Hathaway galvanometer with a natural frequency of about 1000 cps. Note that in the oscillogram it is possible to identify the second harmonic at a frequency twice that of the more intense fundamental

which may be followed by a ventricular contraction after a normal PR interval, may have no accompanying murmur

### MUSICAL MURMURS<sup>2</sup>

Murmurs are musical when to the ear they present a quality which is perhaps best defined by the improved ability to represent the murmur by conventional musical notation. It is easier to place a musical murmur on a musical scale. In the oscillogram, they display regular vibrations at the frequency of the fundamental although occasionally (see Fig 174B also see 1529, Fig 32) the second harmonic is also discernible. In the spectrogram musical murmurs are characterized by the presence of harmonics, a feature which constitutes the most valid and objective definition of musicality (Fig 174)

#### Systolic

Musical systolic murmurs heard over the precordium are of three main types (1) the variety

heard in calcific aortic stenosis (Fig 175a), (2) that of mitral regurgitation and (3) the musical pericardial sound. Each of the three has sufficiently distinctive features that confusion is unlikely

In calcific aortic stenosis one is likely to hear in the aortic area itself and at the base of the neck on the right a noisy non-musical murmur with characteristic diamond or Christmas tree configuration by phonocardiogram. At the left sternal border left midprecordium and apex the murmur may be strikingly musical (161, 221). In the SPCG from these sites there are demonstrated conspicuous chevron shaped harmonics which have their apices at the point in systole where the peak of the noisy murmur is located. It seems likely that the noisy murmur is produced by the jet in the aorta, whereas the musical murmur is related to the regular periodic and therefore musical vibrations of the stenotic valve diaphragm (Fig 176). It is useful to refer to this dissociation

<sup>2</sup> See reference 1083

## MURMURS

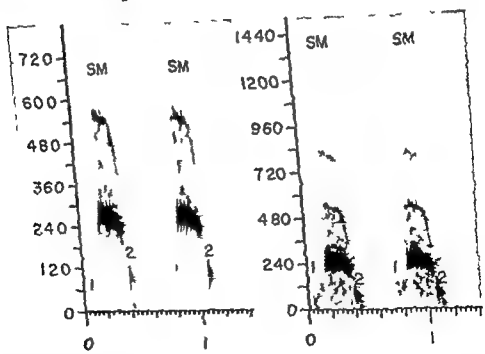
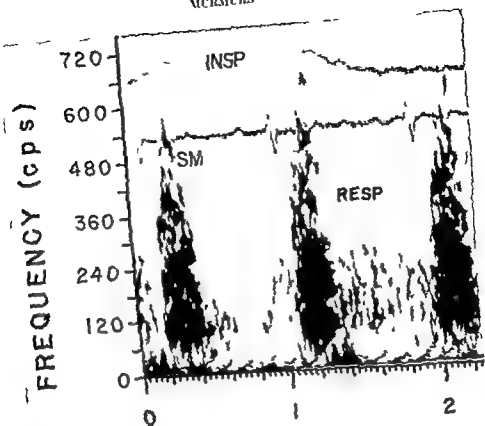


FIG 15 (Above) Systolic murmur of calcific aortic stenosis (pulmonary area). Both musical and noisy elements are present giving a raspy quality to the murmur. The murmur has the Christmas tree shape characteristic of aortic stenosis. (Below) Systolic murmur in calcific aortic valve disease. The murmur was loud and quite musical everywhere and there were no peripheral signs of aortic obstruction. Probably in this case there was fibrosis and calcification of the individual cusps with minimal adhesion at the commissures and very little obstruction to systolic ejection.



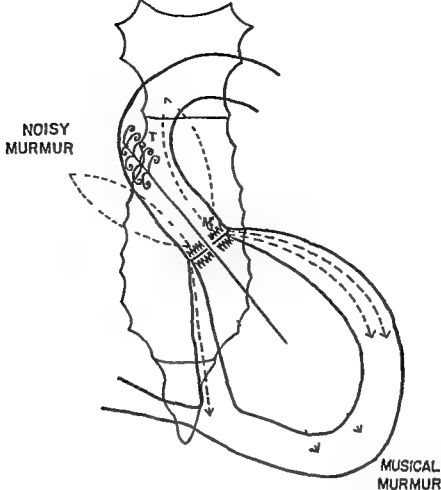


FIG 170 Dissociation of noisiness and musicality in murmur of aortic stenosis—the Gallavardin phenomenon

In aortic stenosis, especially calcific aortic stenosis, the murmur may be musical at the apex and left midprecordium, noisy at the aortic area and base of the neck on the right. The musical component may arise in the stenotic valve and the noisy component higher in the aorta in connection with the jet. The jet noise in other situations is highly directional and zones of maximum intensity such as those indicated by the rabbit ears in dashed lines are theoretically to be expected.

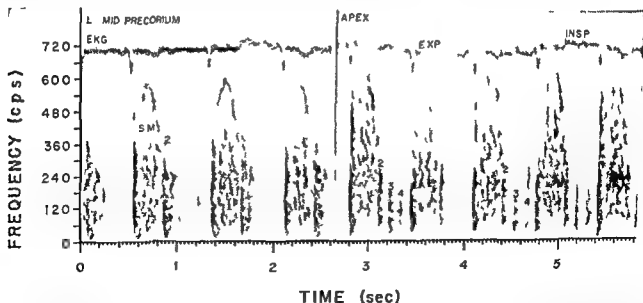


FIG 177 Musical systolic murmur of mitral regurgitation

Left midprecordium and apex in L. L. (610441) 18 year old female who had several attacks of acute rheumatic fever the most recent six years before. She is essentially asymptomatic. The musicality of her systolic murmur is most striking toward the left sternal border. There is a holosystolic murmur which especially in 1 has a harmonic at about 500 cps. At the apex (B) there is a protodiastolic gallop typical of mitral regurgitation and either a pre-systolic gallop or short murmur.

of quality in aortic stenosis in the Calhoun phenomenon.

Frequently the harsh musical systolic murmur best heard at the apex is misinterpreted as being generated at a calcified mitral valve (336 Fig. 3 B). Although such a valve occasionally the cause of a musical systolic murmur, the phonographic and spectrographic pattern are distinct. Specifically the murmur is likely to extend throughout systole as in most murmurs of mitral regurgitation. The harmonic pattern is different as a less specific distinction.

At the apex a delicately musical murmur of mitral regurgitation (Fig. 177) is frequently heard especially (1) during the acute stage of acute rheumatic carditis, (2) in a more permanent sequel of bacterial endocarditis, (3) with a heavily

calcified mitral valve, and (4) in patients with the Mitral syndrome, possibly because of redundant chordae tendineae. The frequency level of the harmonics in this murmur are usually higher than those in other types of musical murmurs. Often, there is only a single harmonic i.e. the fundamental present.

The systolic murmur of ruptured papillary muscle or chordae tendineae described as loud rough harsh and coarse (44) Occasionally (1369) the adjective musical is applied to it that which is not more often done is probably the result of differing ideas of what constitutes musicality.

As a general rule the more obvious a symptom is the more it impresses the mind of the observer and much more importance is attached to it than to less conspicuous phenomena. This is particularly noticeable in

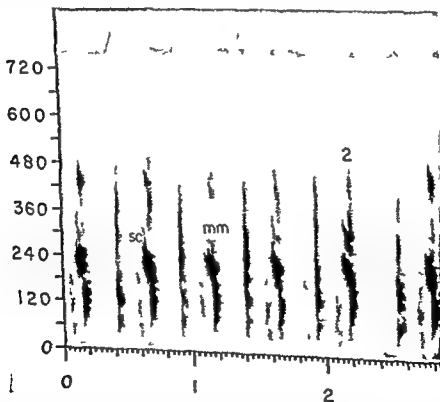


FIG. 18 Musical extracardiac murmur

This musical late systolic murmur developed 18 months before this recording and following a respiratory infection in J. W. (1946) a 6 year-old girl. The murmur was preceded by a systolic click tented to extend over the second sound slightly. The murmur was maximal in inspiration was exaggerated with excitement and exertion to the point of being a little at a distance from the body. It was compared by the parents to the sound of a rusty hinge. It is probably a violin type of pleuropneumonic rub. This patient had mild pectus excavatum which may have contributed to the genesis of the murmur.

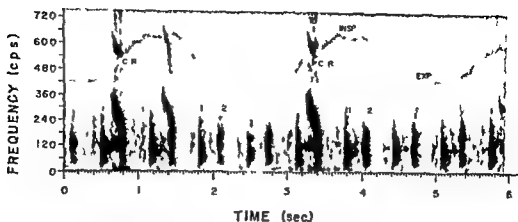


FIG 179 Musical extracardiac murmur late in systole (C R) occurring only in inspiration in patient V W (106004) with cardiomegaly. She had systemic arterial hypertension and multiple pulmonary emboli and probably had had rheumatic fever. This is probably a musical pericardial or pleuropericardial rub of the violin family of musical murmurs. This sound was present for the three years that the patient lived after the recording shown here.

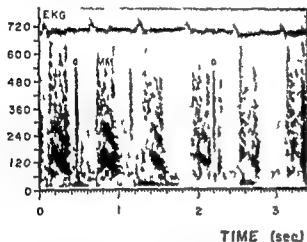


FIG 180 Musical systolic murmur present at the apex and mainly in inspiration in C J (721562) 43 years old during a bout of congestive heart failure of obscure origin.

signs which are detected by auscultation. To the human mind sounds arising from obscure causes have always been a source of mystery, and the human imagination when dealing with the mysterious invariably associates it with something malign.

Sir James Mackenzie 1908

Although one might cite several varieties of murmur to which Mackenzie's comment is applicable it seems especially apropos to the type of musical systolic murmur next to be discussed—the musical pleuropericardial murmur.

A striking musical murmur suggesting a whoop (as in whooping cough) or a squeak as in occasional sequel of pericarditis (Figs 178 to 180). Its mechanism seems to be analogous to that of the violin group of instruments. This sound persists

for years. It is probably more likely to occur when there is pectus excavatum (Fig 178) and/or when the heart is enlarged. It is exaggerated by anything which increases the vigor of the heart beat. Like most musical murmurs which is a class include some of the loudest sounds generated in the cardiovascular system this murmur may be audible at a distance. One mother heard it sounding like a rusty hinge in her six year old daughter after the child had been disciplined. The sound is in late systole is a rule, is often introduced by a systolic click or at times may be replaced by a systolic click, and tends to vary with respiration, being probably most often loudest in inspiration. Either full inspiration or full expiration may eliminate it. In phonocardiograms it can at times be shown that the murmur extends across the second heart sound, feature indicating for its extracardiac origin. The main significance of this sound is its lack of significance. It is easy to ascribe grave significance to it if the phenomenon described is not familiar. A sound audible to the unaided ear at a distance from the subject is likely to have frightening connotations for the patient, his family, and indeed his physician.

Looking back over the medical literature one can find reports of musical murmurs which were almost certainly of extracardiac origin. One of the earliest is that of Maclellan (cited by Magdalen) who in 1813 described a murmur like the bark of a young dog, the valves were found to be normal at autopsy. In 1880 Oler (1168) reported on an apparently normal 12 year old girl who at

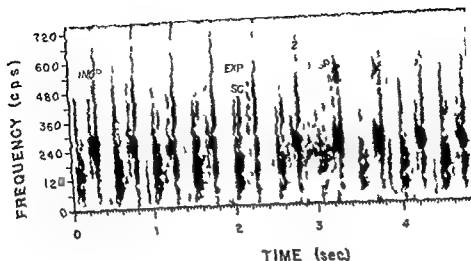


FIG. 181. Murmur extracardiac murmur

Apex in C 5 (B2866) 6-year-old female ten days after onset of acute grippid illness. Note the variability of the late systolic murmur which was most intense in inspiration. There is an intermittent systolic click introducing the murmur. An early systolic click is loudest in expiration. Eight weeks later the murmur was heard only faintly and after vigorous exercise whereas both an early and a late systolic click were now striking.

certain unpredictable times emitted a loud systolic murmur which was distinctly audible at a distance of three feet two inches by measurement and could be heard at any point on the chest and on top of the head. In his *Traité des Maladies du Cœur* Barrié mentioned a 71-year-old man whom he had seen in 1874 and who had a ringing systolic murmur audible four meters from the chest. Having been awakened from sleep by it when it first appeared the patient thought at first there were large flies in the room. The heart valves were later found to be normal.

One of the most detailed analyses of the murmur extracardiac murmur is that of Lian and colleagues (944). They noted two types: (1) the growing functional mid-systolic murmur loudest at the left sternal border and (2) the late systolic murmur introduced by a click, often audible at a distance extending into early diastole, shining in quality, maximal in the sitting position often accompanied by a thrilling (*chatouillement*) which the patient can himself feel. They noted that the latter type could be associated with heart disease and cardiac enlargement and granted that the extracardiac origin can be stated only with some reservations in such cases. They thought that this

type of murmur is usually maximal in expiration and that it is produced by pressure on a trypset of lung with movement of air through narrowed bronchioles. However a later communication suggested that he had revised the latter view (917) and considered the murmur pleuropertic in nature.

In a 33-year-old female patient with primary pulmonary hypertension Levine and Harvey (588, p. 262) described a loud leathery grade VI late systolic murmur which was produced during the Valsalva experiment. The patient heard it herself and found that she could reproduce it at will by holding a deep breath especially if she voluntarily compressed her chest at the same time. It promptly disappeared when the breath was released. Only the usual findings of primary pulmonary hypertension were described at autopsy.

Warburg (1008) described (with phonocardiogram) a murmur similar to that in Figure 180 in a man with mitral stenosis, atrial fibrillation and frequent bouts of congestive heart failure. With an obscure febrile episode the patient noted a noise in the chest which was also evident to the patient's wife. I was able to verify his statement. At every heart beat a clicking or lightly sonorous sound was audible in the room. A phonocardiogram showed that he imitates the sounds

<sup>1</sup> As will be discussed later (p. 944) this murmur may not be of extracardiac origin.

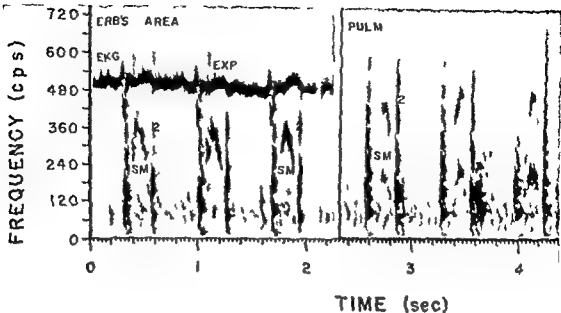


Fig 182

Fig 183

### Musical extracardiac murmur?

L. L. H. (776304) 12 year old boy displayed a musical systolic murmur only after exercise and mainly with a mid respiratory position of the chest. 182, Erb's area after exercise. 183, pulmonic area after exercise with full expiration. L.H.G. omitted for better definition of upper harmonics. Under different circumstances and even from cycle to cycle with what seems the same circumstances there are striking changes in the frequency level of the harmonics. All examinations including cardiac catheterization yielded normal findings. (Trigonoidation at the pulmonary valve i.e., that this is an exaggerated still murmur is an alternative possibility.)

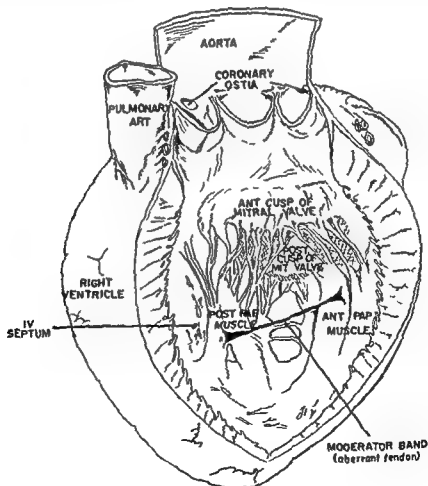


FIG 184 Sketch showing location of aberrant tendon (moderator band) in the left ventricle (After Huchard (718) Rolleston (1308) described a similar specimen)

usually found in the patient there was a new murmur partly during the systole partly immediately after the systole. It was quite clear that the new sound included higher frequencies than the other sound.

Levine and Harvey (888 Fig. 2a) described a 12-year-old woman with rheumatic valvular disease in whom a very peculiar acoustically rough musical murmur was occasionally present in addition to her usual murmur of mitral stenosis and regurgitation and aortic regurgita-

tion. The murmur was usually absent at rest, was always present immediately after brief exercise and disappeared again with rest. It could be heard 1 foot away from the chest. It is known to have been detectable for at least four years and was like a tone in tuning, running over 8. Atrial fibrillation was present.

Weber (161, p. 29) described and illustrated a musical like systolic murmur which occurred in a locomotive engineer who sustained a severe chest contusion in a railroad accident. The murmur was



FIG. 183. Aberrant tendons of ventricle.

The heart in M. V. (12210) colored male who died at the age of 43 years, having been seen in this hospital twenty years previously at which time her heart and lungs were normal. Four years before death she developed toxemia of pregnancy. Thereafter hypertension was persistent and heart failure recurrent. During the period of toxemia and lag in during episodes of heart failure, he was found to have a loud harsh musical systolic murmur accompanied by thrill maximal in the left midprecordial area. She had noted a buzzing sensation. The murmur was widely audible even over the head and sacrum. At autopsy the heart weighed 560 gm. All chambers, especially the left ventricle, were greatly dilated. The aortic valve was normal and no structure was found to account for the musical murmur except a net of aberrant tendons traversing the lower part of the cavity of the left ventricle. The net consisted of a central portion, about 0.5 cm. long in its fixed state, at each end of which were attached two tendons each about 1.2 cm. in length.

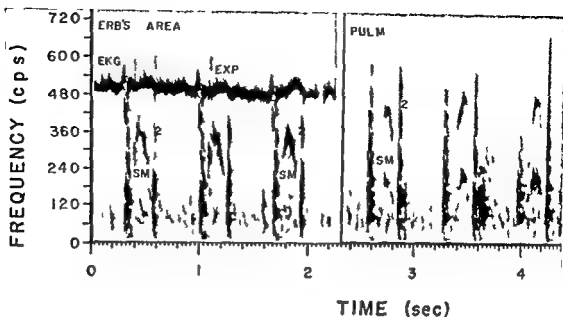


FIG 182

FIG 183

### Musical extracardiac murmur?

I L H (770304) 12 year old boy displayed a musical systolic murmur only after exercise and mainly with a mid respiratory position of the chest 182 Erb's area after exercise 183 pulmonic area after exercise with full expiration LKG omitted for better definition of upper harmonics Under different circumstances and even from cycle to cycle with what seems the same circumstances there are striking changes in the frequency level of the harmonics All examinations including cardiac catheterization yielded normal findings (Trigonoidation at the pulmonary valve i.e. that this is an exaggerated still murmur is an alternative possibility)

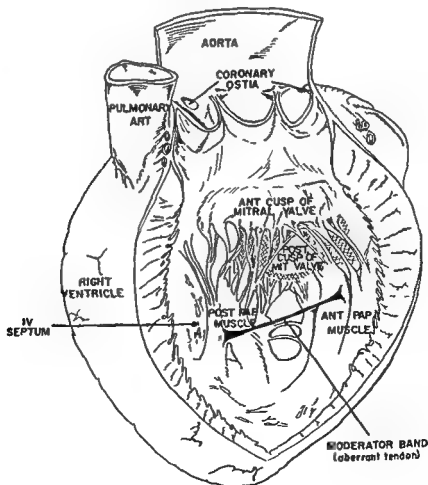


FIG 184 Sketch showing location of aberrant tendon ( moderator band ) in the left ventricle (After Huchard (718) Rolleston (1308) described a similar specimen



FIG. 155 Examples of Chiari nets of the right atrium from Chiari's publication (239)

located in late systole and was often introduced by a systolic click. At times only the click remained.

Rare examples of musical murmur or of musical contribution to noise murmur are produced by aberrant tendons such as those sometimes called "moderator bands" which may cross the stream of flow of blood and be actuated by musical vibration for a murmur comparable to an Aeolian harp. Huchard (713) described the clinical picture associated with aberrant tendon of the ventricle (Fig. 154). The main feature is that the musical murmur is usually not present from birth despite the congenital nature of the aberrant tendon but appears when the ventricle becomes dilated because of some unrelated strain such as systemic hypertension. Only when the aberrant tendon is pulled taut by ventricular dilatation is it possible for a musical tone to be produced. For example Huchard has reported that of a 49-year-old man seen in 1892. Trematosis of severe arterial hypertension, cardiomegaly and heart failure were present. The conventional murmur of mitral regurgitation was heard at the apex and in the left axilla. More medially located maximal in the area of the xiphoid but widely heard especially

to the right of the sternum and up the course of the aorta was a purring or roaring systolic murmur (une sorte de roulement de ronflement de bruit de guimbarde). Unfortunately to the differential diagnosis is the fact that extracardiac musical murmur may occur in the same clinical setting and display the same characteristics although usually more variation with respiration will be demonstrated.

Aberrant tendon of the ventricle is rare. The case presented in Figure 153 is that of a patient seen in the hospital twenty years ago with pathological studies by Dr Arnold H. Pich. It is probable that the Huchard murmur was present in this case.

There is one more common situation in which aberrant tendon may contribute a musical quality to a murmur otherwise not in ventricular septal defects, anomalous tricuspid chordal tendineae may insert at the upper and lower margins of the defect (Fig. 156). These are probably responsible for the harmonics which are occasionally demonstrated by SPCC in the Roger murmur. Probably especially in the common malformation (Fig. 157) the ventricular defect likely to be so related to tricuspid valvular structures that a musical tone is produced.

Closely akin to the musical murmur of moderator band and aberrant chordal tendineae is that

The term was coined in 1931 by King (96) who thought the structure functioned in moderating or checkreining dilatation of the ventricle.





FIG. 186 Aberrant tricuspid chordae tendineae stretching across a right ventricle in aspect of high interventricular septal defect and probably responsible for the musical quality of the resulting murmur. Such cords are often the site of SBI.

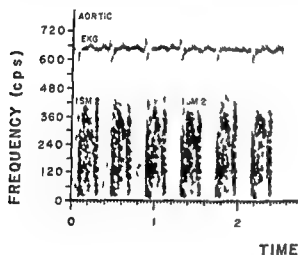


FIG. 187 Sounds typical of most areas of the precordium in D.M.T. (B6145) 3 year old white female with what is thought to be 11 *communis*. There is a striking musical element in the systolic murmur probably caused by anomalous chordae.

loud and widely audible. Atrial fibrillation was present.

Geckeler *et al.* (532) presented spectrograms of a musical systolic murmur referred to by them as

a grunt, which was heard in the second right intercostal space for about ten days after mitral commissurotomy. The author did not think it to be pericardial in origin; however this would appear to me to be the best possibility. Another being that some vibrant structure such as part of the interior (aortic) leaflet of the mitral valve or a chordae tendineae was torn loose and was thrown into vibration with ventricular ejection. The disappearance of the murmur after ten days would be difficult to explain in the latter case.

Taubry and Lebrun (845) described a musical mid-systolic murmur introduced by a click in a patient recovering from lung abscess.

Harvey (652) has described the clinical and phonocardiographic features of what he chooses to term a cardiac whoop. The subject may be perfectly well (one of his cases was a member of the hospital's resident staff) and the sound may be present for an indefinite period. The intensity of the murmur tended to wax and wane with respiration. The vibrations which constituted it were periodic, i.e. musical. The murmur was

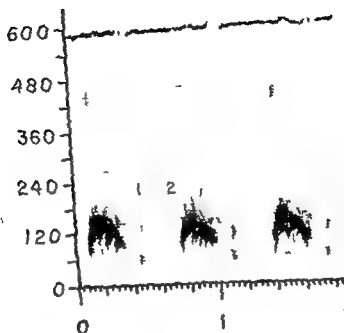


FIG. 199. A diastolic murmur of retroaortic aortic cusp. In this instance it does not being accentuated with atrial systole (as in Fig. 200) the murmur stops abruptly with atrial systole.

of the harmonic in the same general range of frequency as the natural frequency of the thorax will be discussed later (see p. 477) the natural frequency of the male thorax is usually of the order of 120 cps. The fundamental of these murmurs often is in this range and what is conducted best to the cardiac apex. The result is a murmur different in quality through the chest or attenuation of many of the overtones.

The murmur of aortic regurgitation produced by rheumatic fever or by bacterial endocarditis of the aortic valve, such pure murmur usually or such well developed harmonic system. Most often it is a matter of one or two harmonics in the range of an otherwise conventional murmur. Geckeler and his colleagues (32) have presented an exception to this generalization in a patient with pure rheumatic heart disease and a full blown murmur of aortic diastolic murmur. Furthermore, we have had what is probably an identical experience.

In patient P. C. (424089) a very loud blowing diastolic murmur with thrill typical of aortic regurgitation was heard in 1947 when the patient

was admitted for left-sided heart failure. Although there was no history of syphilitic infection or treatment for the same and the serologic tests (including treponemal immobilization test) were all negative syphilis was considered likely. During the following year the aortic diastolic murmur became a conventional one with the usual quality with no very unusual intensities and no thrill. The patient has done quite well on the whole a consideration which with the others mentioned makes rheumatism the probable basis of the original murmur in this patient. The benign course makes syphilitic aortic regurgitation unlikely.

I saw patient P. C. (424089) many times in a murmur previously conventional in nature. In others (cf. Figs. 189 and 192) the murmur element may disappear leaving an aortic regurgitation of the usual noisy quality. The variations in the graphic character of murmur of aortic diastolic murmurs are many and include differences in the following characteristics.

1. The time interval between the beginning of the murmur and the frequency peak.

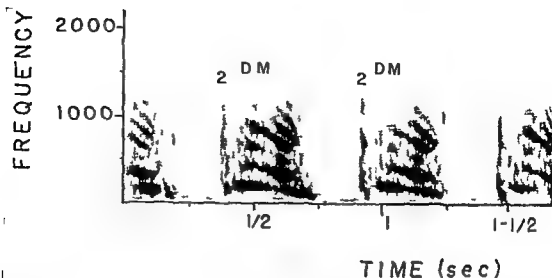


FIG. 189 Retroverted aortic cusp

Typical musical aortic diastolic murmur in patient with syphilitic aortitis. Note intensification of murmur with out rise in frequency level of its harmonics toward end of diastole. Probably atrial systole by displacing the retroverted element into the regurgitant stream, therefore its excitation of vibration without changing its frequency of vibration. When this patient was restudied three years later only a diastolic murmur of conventional noisy quality was found.

which accompanies Chiari's network (Fig. 188) of the right atrium (see p. 410). This is by no means a rare finding, at autopsy 2 to 3 per cent according to Lister (1898), 1 per cent according to Helwig (669). However, it is much less common for a musical murmur to occur in association. In two cases a continuous musical murmur has been described (18) (1871). It is probable that as with ventricular bands, not only does the Chiari network need to be properly oriented in relation to the ventricle, but also dilation of the atrium with tensioning of the network favors development of such a murmur.

### Diastolic

A musical diastolic murmur is rarely heard except in one circumstance—retroversion or some similar deformity of an aortic cusp (82, 537, 606, 1378). The usual cause of retroverted aortic cusp is syphilis, but it has in recent times been recognized that cystic medial necrosis of the aorta of idiopathic type may be accompanied by this accident. Occasionally the aortic diastolic murmur of aortic regurgitation in the Marfan syndrome is musical although more often it is a conventional noisy murmur. A musical aortic diastolic murmur may be heard with bacterial endocarditis and occasionally with the valvulitis

of acute rheumatic fever and with chronic rheumatic valvulitis.

Most features previously described for noisy aortic diastolic murmurs are found in musical aortic diastolic murmurs (Figs. 189 to 191). In addition the murmur of retroverted aortic cusp is in a rule intense. It has the quality of wood, winn, or possibly of the cry of a sea gull or other bird.<sup>\*</sup> It has been compared also to the sound made by a young frog (1392). The ingenious metaphors are legion. More characteristically than in the case of noisy aortic diastolic murmurs, the musical counterpart is *crescendo decrescendo*. The harmonic shows a rise in frequency before the beginning of the decline. This pattern is in large part responsible for the *oo* of the *crescendo* murmur. It is related to an acceleration then a deceleration of the regurgitant stream with corresponding change in the rate at which the retroverted cusp is driven.

There tends to be a change in quality of the murmur of retroverted aortic cusp on transmission to the apex. The basis is a preferential conduction

<sup>\*</sup> Actually experts on bird calls say these sounds resemble those of no birds known to them! Therefore the designations sea gull murmur and cooing dove murmur for members of the musical group may not be appropriate.

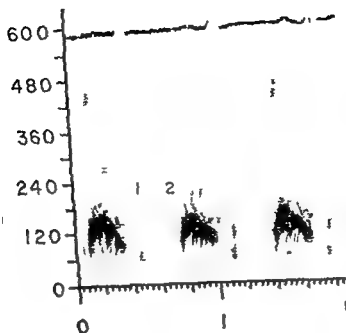


FIG. 190. Murmur diastolic murmur of retroverted aortic valve. In this instance rather than being accentuated with atrial systole (as in Figure 189) the murmur stops abruptly with atrial systole.

of the harmonic in the same general range of frequencies as the natural frequency of the thorax. It will be discussed later (see p. 477) the natural frequency of the male thorax is usually of the order of 120 a.p. The fundamental of these murmurs is often in this range and is what is conducted best to the cardiac apex. The result is a murmur different in quality through the absence or attenuation of many of the overtones.

The murmur of aortic diastolic murmur produced by rheumatic fever or bacterial endocarditis seldom displays such pure musical or such well developed harmonic pattern. Most often it is a matter of one or two harmonics in the midst of an otherwise conventional noisy decrescendo murmur. Cockeler and his colleagues (32) have presented an exception to this generalization, i.e., a patient with presumed rheumatic heart disease and a full blown musical aortic diastolic murmur. Furthermore we have had what is probably an identical experience.

In patient P. C. (124089) a very loud booming diastolic murmur with thrill typical of retroverted a.p. was heard in 1947 when the patient

was admitted for left sided heart failure. Although there was no history of syphilitic infection or treatment for the same and the serologic tests (including treponemal immobilization test) were always negative syphilis was considered likely. During the following year the aortic diastolic murmur became a conventional one with the usual quality with no very unusual intensities and no thrill. The patient has done quite well on the whole a consideration which with the others mentioned makes rheumatism the probable basis of the eagle murmur in this patient. The benign course makes syphilitic as the medical history unlikely.

In some patients (e.g. T. S. 103183) musicality may appear in a murmur previously conventional noisy. In others (cf. Figs. 189 and 192) the musical element may disappear leaving an aortic diastolic murmur of the usual noisy quality. The variations in the graphic character of musical aortic diastolic murmurs are many and include differences in the following characteristics:

1. The time interval between the beginning of the murmur and the frequency peak.

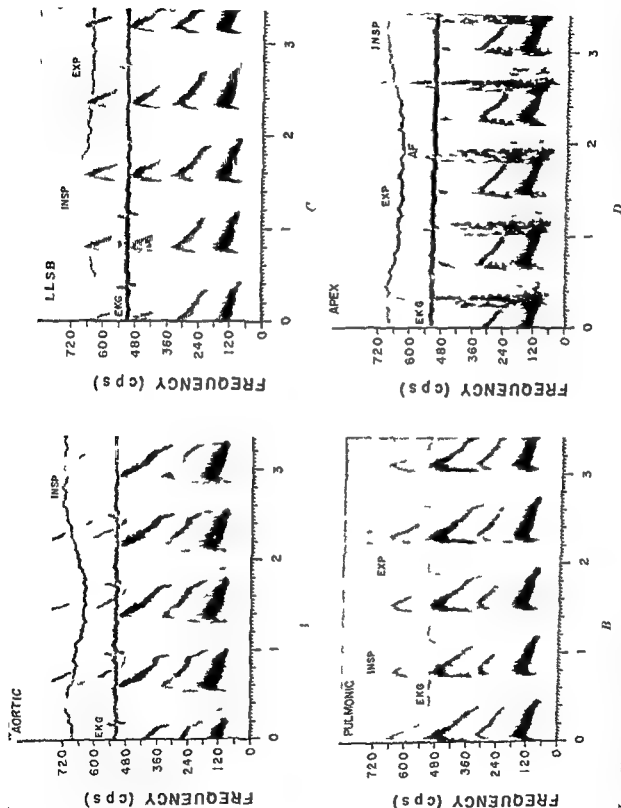


FIG 10] Murmur of retrograde aortic cusp. In this case there is a change in slope of the harmonics with atrial systole. Rather than stopping completely they fall off more rapidly in intensity and frequency. At the apex there is a presystolic murmur which is probably of the nature of an Austin Flint. Its murmurality is puzzling. Another puzzling feature is the attenuation of the second harmonic in the aortic and pulmonary areas. (In a 16 year old patient with a mural aortic diastolic murmur from severe rheumatic fever Gelfand and Bellet (27) Fig. 2) illustrated a murmur of the first element to the murmur.)

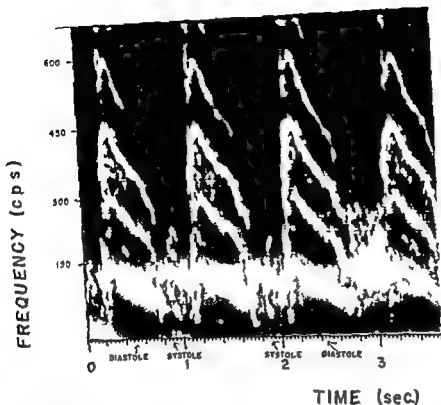


FIG 19 Retroverted aortic cup caused by syphilis in T. S. (10485), 40 year old colored male. Recorded at Philadelphia petrograph made with phase filter. (Compare with Fig 44 in this record the harmonics are not as finely demonstrated as the earlier figure because of halos.) A point of particular interest is the presence of a musical systolic murmur consisting of a single harmonic which is the continuation of the fundamental of the diastolic murmur. Apparently the anomalous valve structure was activated in systole as well as in diastole. When this same patient was studied two and one half years later it was found that the aortic diastolic murmur was now of conventional noisy quality. (See Fig 26a.)

2 The frequency level of the fundamental and other harmonics (cf Figures 191 and 271)

3 The frequency span between the nadir and the zenith of the harmonic

4 The presence or absence of a systolic murmur with similar harmonic continuation (cf Fig. 192, 268 and 272)

5 The degree of cycle-to-cycle variation in the diastolic murmur (see Fig 267)

6 The presence or absence of an abrupt change in the musicality of the diastolic murmur part way through

7 The duration of the musical murmur particularly with regard to interruption by atrial contraction (cf Fig 190)

8 The presence of an increase in intensity without corresponding increase in frequency in

mid-diastole with atrial contraction<sup>8</sup> or just before premature contractions (cf Fig. 189)

9 The alteration in tonality with transmission to the several precordial loci (cf Fig. 10) and 270)

10 The presence of variation with respiration—specifically an intensification early in expiration (cf Fig 267)

I have studied at least one patient in whom the mitral valve seems the likely site for a musical diastolic murmur (cf Fig 194). By means of sulfadiazine the patient was cured of subacute bacterial endocarditis in 1941. Since that time, she has displayed a musical systolic murmur at the apex (caused by mitral regurgitation) and at

<sup>8</sup> Hays and Boggan (638) picture a crescendo in a musical murmur at the time of atrial systole.

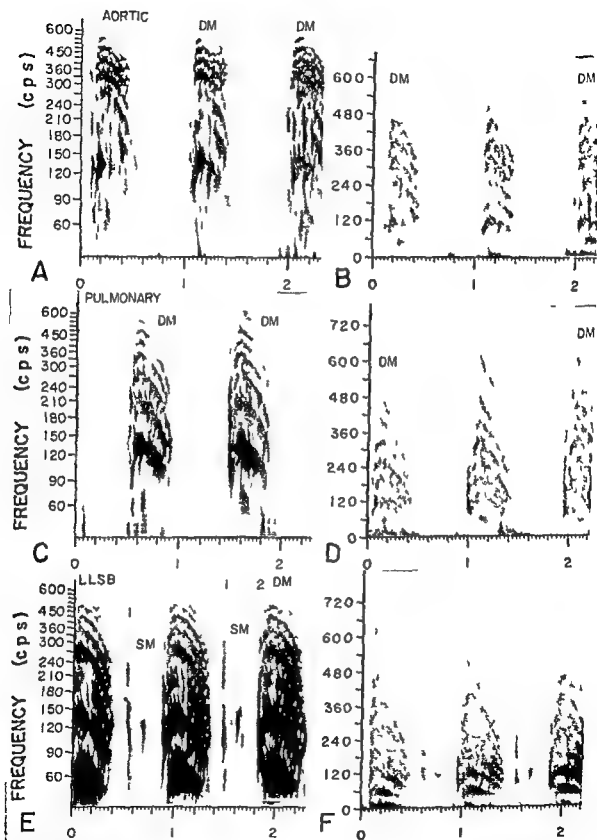


FIG. 19. Logarithmic and linear frequency displays of recordings from aortic (A, B), pulmonary (C, D) and LLSB (E, F) areas in patient with sea gull murmur. Note the evidences of what may be called an absorption in a band of frequency from 150 to 300 cps (aortic are 1). Also note the intensification of the murmur in late diastole without rise in frequency (cf 1A, 183).

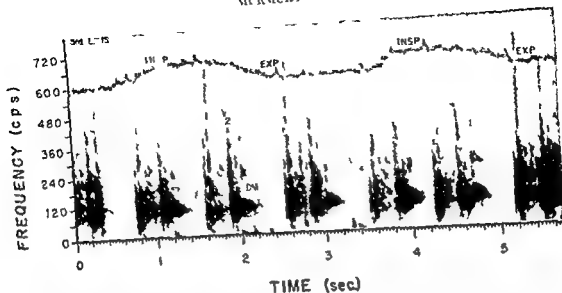


FIG 194 Calcified mitral valve

Third and fourth left intercostal spaces in L B (110613) 67 year old female with advanced rheumatic heart disease and subacute bacterial endocarditis caused by *Streptococcus viridans* and cured by sulfadiazine 14 years previously. There is a mitral late systolic element (better shown in Fig. 316) and a mitral early diastolic. Both are thought to have their origin at the mitral valve. An opening snap was thought to be present in other recordings.

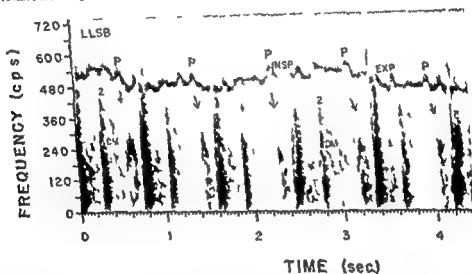


FIG 195 Mitral stenosis sound

Recorded at LLSB two months after mitral valvulotomy for MS in D C (80606) 18 year old female. The PR interval is 0.14 sec. The same mitral atrial murmur with the same IP interval was present four months before operation and has now been observed for a total of nine months. At least two possibilities exist. This may be a mitral presystolic murmur or more likely it may be a mitral atrioventricular murmur. No particular abnormality of the pericardium over the left side of the heart was noted at operation. However, it is possible that contraction of the right atrium is responsible for the murmur. The frequency of the single harmonic varied from time to time being 500 cps in some recordings.

times a mitral quality to the murmur of moderate mitral stenosis. Except for its mitral quality the murmur has the other features of a mitral stenosis murmur. This patient appears then to have a

mitral murmur in both systole and diastole produced at the same generator. In calcific aortic valve disease the distorted orifice may likewise function as a bi-directional generator of a mitral



murmur in both systole and diastole. Occasionally, harmonics are seen in systole in cases of retroverted aortic cusp due to syphilis. In these instances, it is thought that the abnormal cusp pro-

lapses into the aorta in systole, or is at least excited to vibrate during systole.

✓ Musical presystolic murmurs are very uncommon. In the diastolic murmur of retroverted aortic

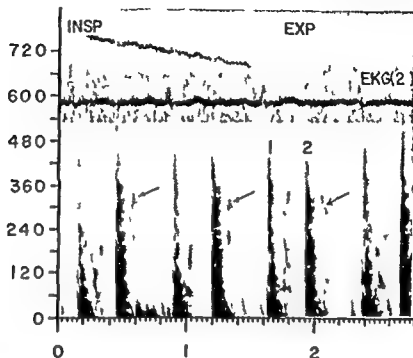


FIG. 106 Musical diastolic murmur of extracardiac origin.

This patient (A. P. 318935) had rheumatic fever in 1919 when 21 years old. The early diastolic squeak indicated by the arrows was heard on annual check ups between 1933 and 1937. There is no evidence of other cardiovascular residual of rheumatic fever. The murmur of mitral aortic regurgitation is usually high pitched but not truly musical in our experience.

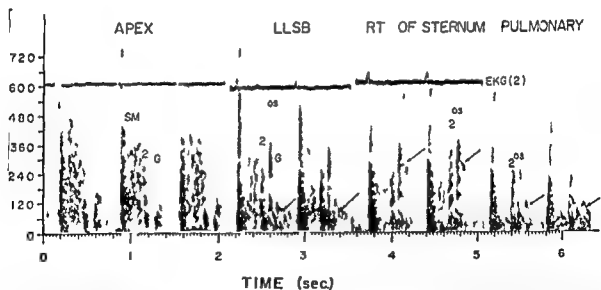


FIG. 107 Musical extracardiac early diastolic murmur (indicated by arrows) in region of lower sternum in patient (S. H. 624689) with rheumatic tricuspid disease and mammoth cardiomegaly. The findings in the mitral area—holosystolic murmur and protodiastolic gallop—are typical of mitral regurgitation. An opening snap 1 percent in the other three records, whether it is tricuspid or mitral cannot be said. This is probably a musical violin type of pleuropericardial rub.

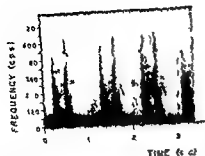


Fig. 134. Mitral extra-systolic early diastolic murmur heard at LLSB for first ten days after valvulotomy for mitral stenosis (66-33). A ventricular premature beat is evident.

cup there may be noted above a mitral presystolic accentuation. Cilo (220) described a patient with a phallic aortic and heart failure, aortic regurgitation and conventionally not a early diastolic murmur and in the aortic area only a presystolic puffy sound.

Figure 19) present a mitral presystolic murmur in mitral stenosis. It is entirely possible that this murmur was pericardial in origin. However, if it is present before a valve after operation and no particular evidence of pericarditis over the left atrium was discovered at operation nor was the anatomy of the mitral valve unusual. Friedman and colleagues (944) had a case they interpreted as an extra-systolic presystolic murmur but it was too early to permit phonocardiographic confirmation.

On rare occasions mitral diastolic murmur can be extra-systolic in origin (716, 945, 946). I have seen such in the first ten days following cardiac surgery and in large rheumatic heart which in addition to impinging prominently on anterior thoracic wall structure may have pericardial roughening (Fig. 197). In one patient a faint early diastolic murmur which was absolutely the only residuum of rheumatic cardiac persisted for three years (Fig. 196) but had disappeared after five years. After cardiectomy for valvulotomy or aortic repair a mitral diastolic murmur may be present for several days. It may be early diastolic or aortic-systolic. It occurs especially often in rheumatic heart after mitral valvulotomy (Fig. 195).

## Other Mitral Murmurs

The possibility of a mitral cardiopulmonary murmur representing an essence of pulmonary rise (690) suggested by Lacombe (see p. 9). It might occur in either a systole or diastole. I have not concerned myself of this mechanism in any specific case. In most cases in which this is a possible pleuropulmonary rubbing seems more likely.

The mitral murmur associated with tricuspid valve thrombosis of the atrium and the ectopic regurgitation of the mitral valve is common. The mitral aortic cleft and the mitral murmur arising in narrowed aortic are of considerable interest and some practical importance. Mitral murmur of incompletely understood mechanism occur with tricuspid regurgitation (p. 420).

In one patient in whom the right atrium was occupied by a ball valve thrombus (I. C. J. H. H. 646029) and in a second with the left atrium in the site (H. K. J. H. H. 94208) a loud mitral systolic murmur was heard. A patient (H. S. 94271) with thrombosis of the left atrium had a similar murmur. Unfortunately recordings were not available in any of the patients. From the findings of post-mortem examination in the second and third and the findings of operation in the first it seems likely that tricuspid or mitral regurgitation created by the return of the ball in holding apart the margins of the valve is possible. This is of the murmur. Immediately to mind comes an analogy to the aortic in which a ball by rapidly ejected occlusion of an orifice modulates a stream of air to produce a mitral sound.

In the case of involvement of the right atrium there was a high pitched systolic sound heard at the left lower sternal border and at the very end of inspiration for the first three or four beats after the breath was held.

In the case of left atrial thrombus the murmur was likewise highly changeable. During deep expiration there was a very harsh screaming or grunting systolic murmur immediately following the first heart sound. The murmur could be heard with the stethoscope one centimeter from the chest wall. At full inspiration this high pitched sound completely disappeared. Position did not affect the murmur however the mitral systolic murmur completely disappeared for a few beats at full inspiration. There appeared to be an ejection

\* See ref. 1212 for a somewhat similar case.

murmur in both systole and diastole. Occasionally, harmonics are seen in systole in cases of retroverted aortic cusp due to syphilis. In the e in stances, it is thought that the abnormal cusp pro

lapses into the aorta in systole, or is at least incited to vibrate during systole. Musical presystolic murmurs are very uncommon. In the diastolic murmur of retroverted aortic

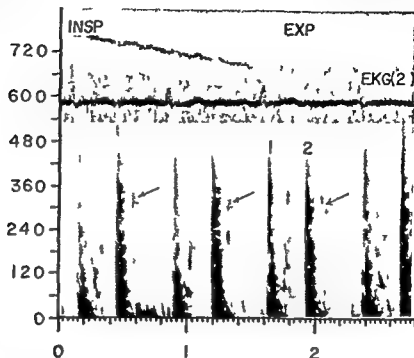


FIG 196 Musical diastolic murmur of extracardiac origin

This patient (A 1 345935) had rheumatic fever in 1949 when 21 years old. The early diastolic squeak indicated by the arrow was heard on annual check ups between 1953 and 1957. There is no evidence of other cardiovascular residua of rheumatic fever. The murmur of minimal aortic regurgitation is usually high pitched but not truly musical in our experience.

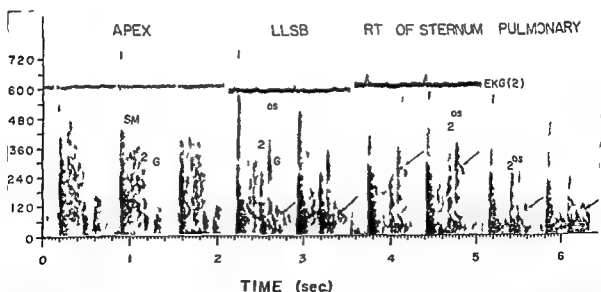


FIG 197 Musical extracardiac early diastolic murmur (indicated by arrows) in region of lower sternum in patient (S H 62689) with rheumatic fever in 1949 when 21 years old. The findings in the mitral area—holosystolic murmur and protodiastolic gallop—are typical of mitral regurgitation. An opening snap is present in the other three records whether it is tricuspid or mitral cannot be said. This is probably a musical violin type of pleuropneumal rub.

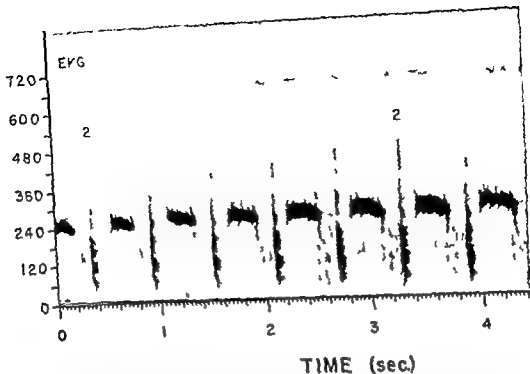


FIG. 19. Murmur venous hum

Recorded at the base of the neck on the right in L. A. K. (79233) 17 year old girl with coarctation of the aorta. A murmur first occurred consistently in late diastole & was heard in the neck with unusual clarity and was caused by hypertension. Note the gap between & and the beginning of the murmur (this was evidence suggesting that venous flow was responsible for the murmur since it might be expected that a gap of approximately this duration would be necessary for opening of the tricuspid valve and buildup of venous flow. For no apparent reason in the course of the recording the murmur's beam lowered with slight increase in pitch. It also became slightly resonant on beginning diastole & there would appear to have been an acceleration of venous flow.

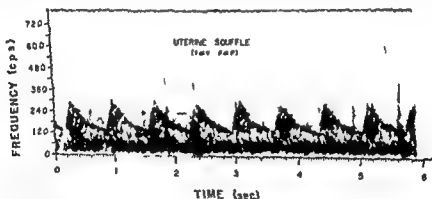


FIG. 20. Murmur uterine souffle with shape of arterial pulse pressure curve. The contour of the single harmonic which is its most striking feature is determined by the relation of proportionalities linking frequency to arterial pressure (see text).

and this is displayed in Figure 208—a slightly little tune is produced with at least three notes resulting from three levels of venous flow, related to atrial & to ventricular systole and ventricular diastole.

The uterine souffle is usually noisy but occasionally may have a conspicuous musical character. (Most of the murmur murmurs discussed in this section are rarer counterparts of more commonly occurring noisy murmurs.) The musical

after a few beats and with post inspiratory apnea the murmur gradually returned, becoming louder and louder with each beat. It is interesting and diagnostically important that respiration had converse effects in the two cases. The usual experience is that tricuspid murmurs are accentuated by inspiration, presumably because of increased venous return to the right side of the heart. On the other hand, venous return to the left side probably falls, as a rule with inspiration and is increased with expiration. Mitral murmurs may show variations with respiration paralleling those in venous return to the left atrium.

In the case of anatomy of the left atrium the systolic murmur was less noticeably variable. It was described as follows. At the apex transmitted poorly to the axilla and extending over along the left sternal border there was a loud high pitched cooing type Grade IV systolic murmur masking the first sound. Weinstein and Arata (1519) described a harsh systolic murmur in a case of anoma of the left atrium. Black, Parker and Edwards (116-408) described a very high pitched whistling, musical systolic murmur. Ciron (237) and Hoffmann (698) described a raspy systolic murmur. However in none of the literature on bill valve thrombus of the atrium (443) have I found reference to a murmur of the type described above.

Occasionally the murmur of ruptured chordae tendinae or ruptured papillary muscle may be partly or largely musical in quality. Butterworth (203) provided me with tape recordings made on repeated occasions over a period of several months in what was probably ruptured papillary muscle from coronary artery disease. The pattern of musicality of the systolic murmur varied somewhat during the period of observation.

→ In dissecting aneurysm of the aorta a musical murmur developing abruptly over the upper thorax, usually in the first and second interspace or at the base of the neck, can be a helpful diagnostic clue. I have had experience with three varieties of musical murmur associated with dissecting aneurysm. In one group a systolic murmur appeared with the dissection and was explicable, seemingly on the basis of vibrations in the lip created by the intimal rent or in the fibrous cords which usually traverse the false channel (1072 p 52).

In another group, the murmur was likewise systolic, was limited sharply to the base of the neck, and appeared to be generated in a partially occluded innominate artery or other branch arising from the arch of the aorta (Fig 467). In these patients the mechanism appears to be identical to that operating in other cases of musical murmurs with partially occluded arteries (see later).

In a third group, represented in my experience by a single patient, a bizarre "bull fiddle" sounding late diastolic murmur occurred, loudest in the left first and second interspaces and audible only with longer diastolic periods such as in the compensatory pause following a premature contraction. One of the two mechanisms already mentioned was possibly operating in this patient whose murmur and anatomical findings are demonstrated in Fig. 9 p 22 of reference 1071. Furthermore the proximal diverticulum in the vicinity of the aortic valve, with accompanying lip, may have had something to do with the murmur. Finally, it is possible that the murmur may have been generated at the aortic valve. Indeed the patient had in addition a conventional non-early diastolic decrescendo murmur. It is possible that near the end of a long diastole circumstances were such that the aortic valve that the musical murmur occurred.

Roberts (1284) described a 33 year old man with dissecting aneurysm in whom he found a very intense systolic thrill of a peculiar vibratory nature and a long coarse whistling systolic murmur over the aortic area and neck vessels. As is shown clearly in his drawing, autopsy revealed just above the right anterior coronary cusp a lip which had been dissected up in the process of burrowing back in the vicinity of the aortic ring. This lip was certainly in a position to vibrate musically during ventricular ejection and was the only structure found to account for the systolic thrill and murmur described.

→ Venous hums may be spontaneously musical or may become musical with a certain amount of pressure on the stethoscope or microphone. It is usually difficult to reproduce the musicality with any predictability. Figures 199 and 208 display two examples of musical venous hum. The loudest and highest pitched component is likely to be in diastole because venous flow is most rapid at this time in the cardiac cycle (Fig 199). Sometimes—

uterine souffle (Fig. 200) is composed of a single prominent harmonic which describes an arterial pulse pressure curve. The frequency at which the generator—the tortuous dilated uterine arteries—varying in unwontedly large volume of blood because of the pregnant state of the uterus—is driven is proportional to the velocity of flow which in turn is proportional to the volume of flow which finally is proportional to pressure. Thus, frequency at any one moment is proportional to pressure.

Artificially occluded arteries characteristically produce a noisy murmur which is related to ventricular systole with its peak in recording removed from the QRS of the multitraceously displayed electrocardiogram by an amount dependent on the distance from the heart to the obstructed vessel. Occasionally the murmur may be musical and may be continuous; both characteristics require special explanation. Myers and colleagues (1136) noted that the murmur of arterial obstruction can be continuous rather than only systolic when the intra-arterial pressure beyond the obstruction is so low that the pressure proximally exceeds it at all times during the cardiac cycle. Much has not been done to understand the mechanism; probably comparable to that in bronchial asthma (1079). That the murmur shown in Figures 201 and 202 has the contour of an arterial pressure pulse has the same explanation as is provided for the musical uterine souffle

(see above). The murmur was recorded over the left axillary bifurcation in 2 men with severe generalized atherosclerosis on the basis of hypercholesterolemia.

The recording in Figure 387 represents another situation in which there may occur a musical murmur with distinct analogies to uterine souffle and which represents in effect relative stenosis of arteries. The patient has tetralogy of Fallot with pulmonary stenosis. This is so-called pulmonary arteriovenous since the blood supply of the lung is from the aorta via bronchial arteries. The low pressure in the system which these arteries supply accounts adequately for the continuous nature of the murmur and in a similar mechanism for the fact that the contour of the fundamental is that of an arterial pulse pressure curve.

It will be clear from the above discussion that hemodynamic factors are clearly reflected in the shape of the harmonics of musical murmurs. This fact is illustrated by the murmur of calcific aortic stenosis, retroverted aortic cup and by venous hum and the various valvular murmurs with the pattern of an arterial pulse pressure curve.

## EXTRACARDIAC MURMURS

### Pericardial Friction Rubs

Pericardial friction rubs (Fig. 203) are really murmurs usually of the noisy variety occurring usually but not necessarily invariably in both

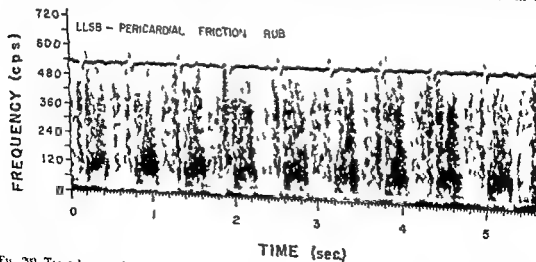


FIG. 203. Typical pericardial friction rub in a patient with acute tuberculous pericarditis. Note the rather diffuse frequency composition. Accentuation of the friction sound in ventricular systole—early diastole and a third

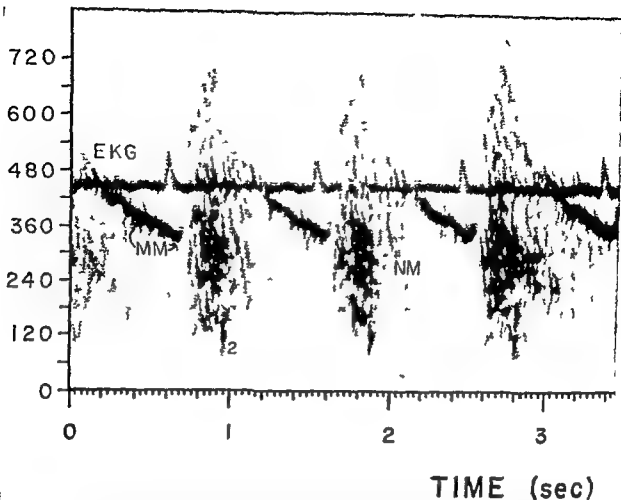


FIG 201 Musical murmur of partial arterial occlusion

Recorded over left carotid bifurcation in J. I. P. (184964) 49 year old male with idiopathic hypercholesterolemia. There is a noisy murmur with its peak at about the same time as the second heart sound which is faintly visible (2). There is a continuous pure tone with changeable frequency level describing an arterial pulse pressure curve. Continuous murmurs like that shown here and in the next figure are important in the early diagnosis of carotid artery insufficiency (3171).

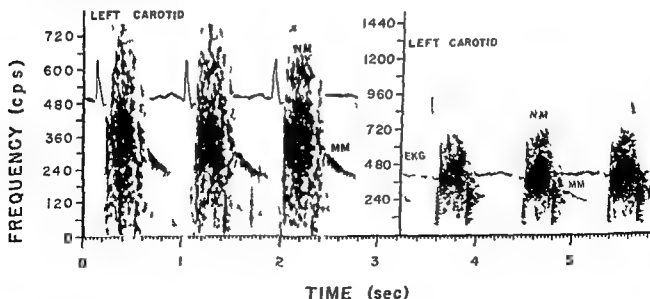


FIG 202 Partial arterial occlusion

Recorded over left carotid artery of C. L. (781887) 62 year old man with severe generalized arteriosclerosis. Displayed on 720 and 1440 cps scales. The murmur at the peak of systolic flow (NM) is largely noisy (with submerged harmonics). It is followed by a musical murmur.

Structural murmurs which appear to have their origin in pericardial roughening are discussed on pp. 138 and 204. Bourne (131) described an extremely normal recruit who displayed a buffing murmur which corresponded with all of the heart's movements—a tole and diatole of the ventricles and a tole of the trachea—producing a triple buffle sound. (It was eliminated by inspiration and was only heard during the last half of expiration and the beginning of inspiration.)

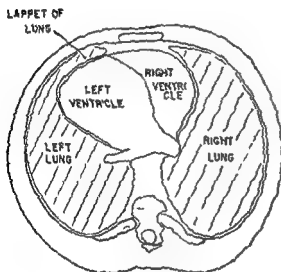


FIG. 203 The postulated mechanism of the cardiopulmonary murmur

tion. This was presumably a pleuroparietal friction which happened to be detected at the time of the examination of a person with subclinical pleurisy. However it is possible that the phenomenon was of long standing character. It is a pity that circumstances did not permit follow up.

### Cardiopulmonary Murmurs

As Jenner pointed out the movement of air in lung tissue through pressure in the contracting lungs is likely by the distorting heart on a lappet of lung to produce origin of some murmurs (11, 20). It is to these that the term 'cardiopulmonary' is most legitimately assigned. Often it is difficult to cite origin of murmurs further than extracardiac and the differentiation of pericardial (or pleuroparietal) and cardiopulmonary origin is impossible. See page 15 for Hopk's entertaining description of cardiopulmonary murmurs in two young men who wore tight waistcoats.

Characteristically cardiopulmonary murmurs vary with respiration. Some are louder at or present only in inspiration (the inspiration rhythm syndrome of Taquet and Nave (1470)) some in expiration. The murmur is usually a tole. In fact I have not encountered a diastolic murmur I could feel confident was cardiopulmonary (31, 118) although diastolic murmurs which appear to be of pleuroparietal origin are familiar from experience in several cases. The part of a tole

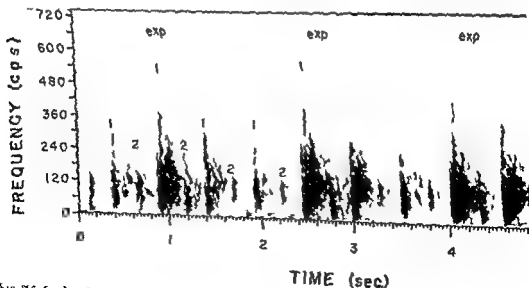


FIG. 211 Cardiopulmonary murmur at apex in patient with marked a/cute. There is a decreasing systolic murmur present only in expiration—probably in essence a vesicular breath sound (see p. 138)



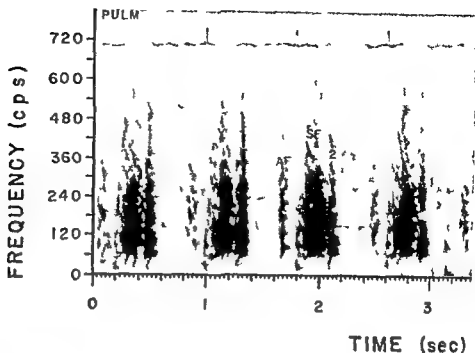


FIG. 204 Atrial friction and circumscribed systolic friction in I M (618231) 62 year old female with recurrent pericarditis of obscure cause. Background noise throughout record.

systolic and diastolic, and having a characteristic superficial quality variously described as leathery, scratchy, etc. Pericardial friction rubs are notorious for their variability of location and intensity from time to time. There is no usual place of maximum audibility. In fact, when pericarditis is suspected, it is important to listen frequently in all parts of the precordium and with the patient in various positions.

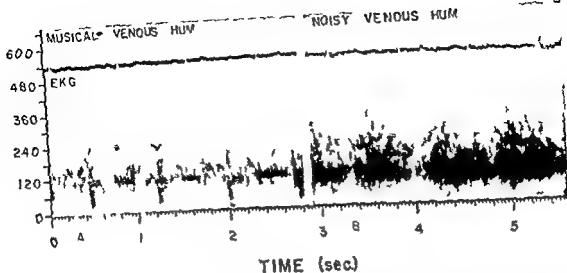
Pericardial friction rubs may, during their evolution or devolution, be limited to systole or much more rarely to diastole. In full blown form, they have three components (301) systolic, protodiastolic, and presystolic (atrial). This may give a rufroid trill (choo choo choo) rhythm to the sound.

At times a pericardial friction rub may be mistaken for the systolic and diastolic murmurs of aortic valve disease. Pressure on the precordium with the stethoscope is a time honored method for accentuating a pericardial friction sound and differentiating it from an endocardial murmur. The mechanism of the accentuation is probably acoustic filtration although closer apposition of the rubbing surfaces may play a role in the phenomenon especially in children with pliant chest walls. Ribaud *et al.* (1268) questioned the specificity of this sign and pointed out that endocardial murmurs may be accentuated by the same maneuver.

In the oscillogram there is little or no basis for differentiating a pericardial friction rub from an endocardial murmur. In the spectrogram the quality of the pericardial friction rub is represented by the more diffuse frequency content, i.e., the more uniform distribution of energy over an appreciable range of frequency. On the other hand, endocardial murmurs as a rule tend to have a kind of dominant frequency, albeit wide, with much sharper fall off above and below this level.

There is usually a silent gap between the atrial component of a pericardial friction murmur and the first heart sound (Fig. 204). This feature with a PR interval of normal duration helps differentiate a presystolic friction from a presystolic murmur, for example, patients with mitral stenosis who have had recent a thorotomy.

Musical pericardial friction rubs produced by a mechanism comparable to that of the violin family of instruments have been commented on (p. 206). They are frequently a long standing manifestation. Occasionally in acute pericarditis the murmur may be musical, resembling to the ear the rubbing of a wet finger on glass. Furthermore for weeks after recovery from acute pericarditis the patient may show a grunting or creaking systolic murmur which because of its subsequent disappearance is almost certainly of pericardial origin (14, 230).



Figs 205 and 206 Venous Hum

Record 1 from the right side of the neck in a normal 33 year old female (C F M) in the sitting position. The murmur at time is musical at time noisy. In any one cardiac cycle there are several changes in pitch of the musical murmur resulting in a pretty little tune. When auscultating the murmur has maximal intensity in diastole.

musical velocity of flow in the veins at the base of the neck. The characteristic diastolic venous hum (which may occur in thyrotoxicosis) from a thyroid bruit (p 445) which is in essence an arteriovenous fistula and, as in all arteriovenous fistulae, has its maximum intensity related to ventricular systole albeit late systole. Occasionally a venous hum is interrupted and displays at least two separate components, one in systole one in early diastole. This is most likely to occur with musical venous hums (Fig 201) but noisy hums may also display this feature (Fig 464). Valon and Kramer (1143) measured inferior vena caval flow and found two maxima, one during systole and one during the first part of diastole. Atrial systole produced a reduction in the volume and velocity of flow. Hollidark and Wolf (706, p 138) illustrated a case of second degree heart block with a venous hum which was interrupted at the time of each atrial systole.

In 1937 Linn (916) described in two patients a continuous murmur in the right intercostal-vertebral space. A small tumor of the lung was present in both and responsible for the murmur in the opinion of the author. He thought furthermore that the murmur was of the nature of a venous hum caused by compression of pulmonary veins.

By older writers (618, p 312) we are informed

that a venous hum can be heard over the femoral vein in cases of anemia and that it is accelerated by raising the leg. Possibly both accelerated blood flow and constriction of the veins at the inguinal ligament are factors in the latter effect.

### The Carotid Bruit

The carotid bruit (referred to by Hollidark and Wolf (706) as the autochthonous carotid murmur) is distinguished from the transmitted murmur of aortic stenosis by a poorly recognized phenomenon with practical significance. Lecomte, Bouillaud and many others in the first 30 years or more of thoracic auscultation considered what we now call the venous hum to have its origin in the carotid artery, presumably through compression of the artery by skeletal muscles in its vicinity. The protagonists for the venous origin of this sound did such a thorough job that all ideas of the origin of murmurs in the carotids were discarded. Although compression by surrounding muscles is unlikely, there are several circumstances in children in particular which can give rise to a murmur at the base of the neck. Firstly, the bifurcation of the innominate artery into the right carotid and right subclavian arteries receives a direct blast of blood from the ventricle. Secondly, there is an edge at this point which splits the stream of blood. In children with familiarly

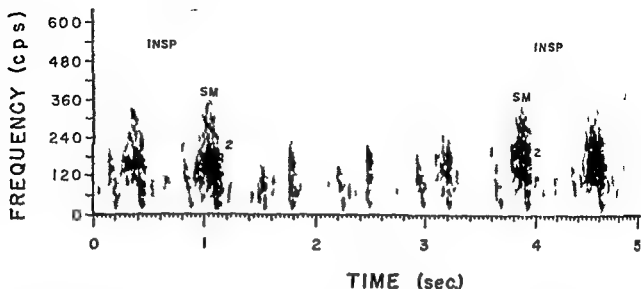


Fig. 207. Cardiopulmonary murmur at apex in patient (P. R. 361900) with flat chest and cardiomegaly caused by hypertension. A crescendo murmur occurs in systole. Mechanism probably is in Fig. 1.

occupied by the murmur varies. For example, in Figures 206 and 207 are presented two instances of cardiopulmonary murmur: one is decrescendo and early systolic occurring mainly in expiration, the other is crescendo and late systolic occurring mainly in inspiration.

Hyzer (1469) claimed that these murmurs may be heard with great intensity in the back over the left lung.

It is theoretically possible for a cardiopulmonary murmur to be musical as a result of the movement of air through bronchioles narrowed by some other mechanism, the whole situation being comparable to that in bronchial asthma. Culo (218) described the case of a 'chronic bronchitic' in which he postulated this mechanism. In this case, furthermore, the murmur was exceedingly intense and was diastolic in timing. Tim and colleagues (944) applied the same explanation for certain systolic and diastolic musical murmurs. Usually a pleuroperecudial violin-like mechanism for musical extracardiac murmurs seems, on the basis of circumstantial evidence, more likely than a cardiopulmonary origin.

### VASCULAR MURMURS

#### Thoracic Arteriovenous Fistulas

Thoracic arteriovenous fistulas will be discussed in connection with the conditions simulating patent ductus arteriosus (p. 403).

#### Venous Hum

Venous hum (838) is because of its frequency and because of its possible confusion with lesions

of grave significance, one of the major cultural phenomena (Figs. 208-209). It is usually maximally audible at the base of the neck, especially at the right. It may be audible in fact loud over the upper part of the chest in young children, suggesting some lesion of grave significance. For example, in fourteen children of ages 3 to 14 years, cardiac consultation was sought because of a mistaken diagnosis of patent ductus in three and of aortic regurgitation in two (1178). The abrupt enlargement of the veins with confluence of several streams may be responsible for the usual localization of venous hum. It occurs most often and in most striking form in children in the upright position and in inspiration—all conditions associated with increase in velocity of flow. In girls it usually persists until later in life than in boys. A venous hum can often be elicited by having the subject turn his head away from the side of auscultation, thereby putting the veins on the stretch or otherwise narrowing them, and can be abolished by light pressure on the veins above the point of auscultation. It is more frequent in menia; the reduced viscosity of blood is probably responsible. Like most murmurs, it is exaggerated by fever. It also may be increased by thyrotoxicosis. Venous hum may occur more frequently with coarctation of the aorta, possibly because of increased flow in the part of the body proximal to the coarctation.

The venous hum is most often a continuous murmur which has its maximum intensity in ventricular diastole (Fig. 209) at the time of max-

the neck on the right may occasion concern about the possibility of mild aortic stenosis (Fig 210). If the murmur is heard only at the base of the neck and not in the aortic area it is probably a carotid bruit and of no serious prognostic significance.

The carotid bruit occurs in a situation with aortic regurgitation (Fig. 211) because of the rapid ejection of a large stroke volume. A systolic murmur at the base of the neck on the right need not indicate a occluded aortic stenosis.

### Peripheral Vascular Murmurs

Peripheral vascular murmurs (407-703) include, of course the murmur of arteriovenous fistulas and other fistulous lesion (406) of partial arterial occlusion, and of arterial collaterals. Certain special categories such as uterine souffle, mammary souffle, the Crivellier-Bismuth murmur and cephalic bruit will be discussed separately.

In arteriovenous fistulas (Figs. 212 to 214) the murmur is characteristically continuous with its peak in late systole and delayed by an interval dependent on the distance of the fistula from the heart. The intensity of the murmur can be reduced by pressure on either the affluent artery or the effluent vein, although pressure on the former is more effective in obliterating the murmur. Slowing of the heart (Brithman's sign) usually accompanies obliteration of the fistula. The murmur is essentially the same whether the fistula is of traumatic origin (Fig. 212) represent a congenital malformation (Fig. 213 to 214) or is a functional fistula in a toxic goiter (Fig. 215), a highly vascular neoplasm such as the metastases of chorionepithelioma (Fig. 217) the bone lesions of Paget's disease, a pregnant or monstrosity uterus or even lactating breasts.

Bonan and Calo (127) found the typical murmur in an arteriovenous fistula of the forehead

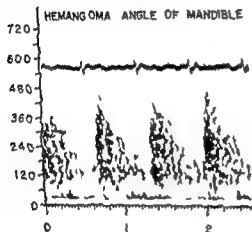


Fig. 1 Congenital fistula

Recorded at angle of mandible on left in J. T. (30) 40-40 year old female

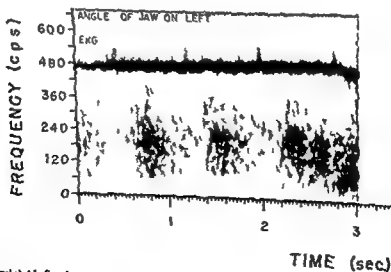


Fig. 13 Congenital fistula at angle of jaw on left in R. B. (401) 53 year old white male. An incomplete operation was performed seven years previously. There is a cystic area in the mandible. Although the murmur is not a typical type of an arterial pulse pressure curve.

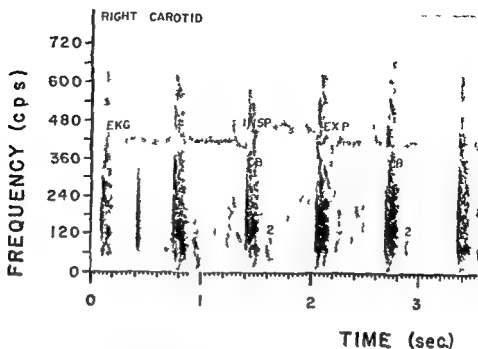


FIG. 210 Carotid bruit

Base of neck on right in B. A. J. (A68968) 14 year old female who had acute rheumatic fever at the age of 5 years. There is no definite evidence of residual valvular damage. The circumscribed sound in the neck probably is generated locally in the innominate carotid subclavian axis since its onset is appropriately late and there is no murmur in the aortic area. At the apex there was a teleostolic click probably caused by pericardial adhesion.

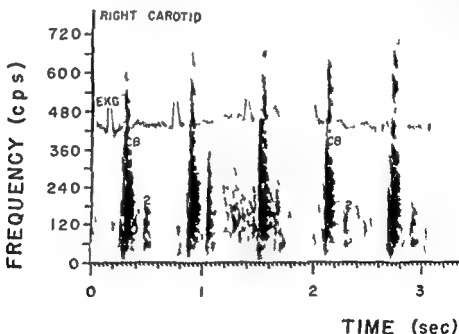


FIG. 211 Carotid bruit

P. S. (24750) 32 year old female has aortic regurgitation but no impressive murmur of aortic stenosis in the aortic area.

vigorous circulations. Circumstances for the production of a murmur at the innominate carotid subclavian junction are likely to exist at the peak of systole (Fig. 210) and to be favored further if

fever and anemia (706, p. 111) are present. In children who are being followed for evidence of cardiac involvement after acute rheumatic fever the presence of a systolic murmur at the base of

## MURMURS

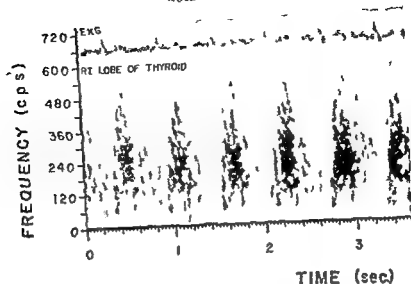


Fig 216 Thyroid bruit in E.D. (393632) 46 year old female. The murmur is maximal in systole unlike a venous hum which is usually maximal in diastole. See Figure 460 for the German Means service in this patient.

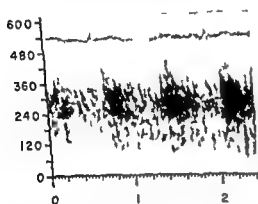


Fig 217A Arterial souffle

Murmur produced in a cuticular meta (a) of choriocarcinoma in retroperitoneal area of upper abdomen and audible over most of the abdomen and lower back. At operation uncontrollable bleeding was encountered and the patient (P.G. 393636) 26 years old died. The top of the murmur has the shape of an arterial pulse pressure curve.

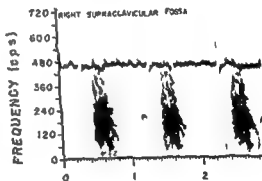


Fig 217B Atherosclerotic stenosis of right subclavian artery

Recorded in right supraclavicular fossa of I.L. (39394) 27 year old male with severe hypertension and advanced premature atherosclerosis. The murmur is distinctly musical. After the application of heat to the right arm (shown here) the murmur became longer extending somewhat into diastole. The principle of treatment used by Myers and colleagues (1136) is well illustrated.

incident to bleeding with leech. Figure 217 presents a case of arteriovenous fistula at the same site secondary to trauma with skull fracture.

Malmer (1026) described a 12 year old boy with a hemangioma in the left upper quadrant of the anterior abdominal wall producing a murmur which was well heard over the heart and led to a mistaken diagnosis of congenital heart disease. Firm pressure on the tumor which felt like a bag of worms abolished the murmur.

Renal arteriovenous fistula a rare occurrence has in all reported instances been accompanied by a loud continuous murmur (627A). A congenital aneurysm with subsequent rupture may be the mechanism in some cases. Hypertension is usually present and combined with a murmur in the proper site makes the diagnosis.

In Paget's disease of bone vascular lesions functionally equivalent to AV fistulas develop in the involved areas of the skeleton. The cardiac output may be raised and the oxygen content of

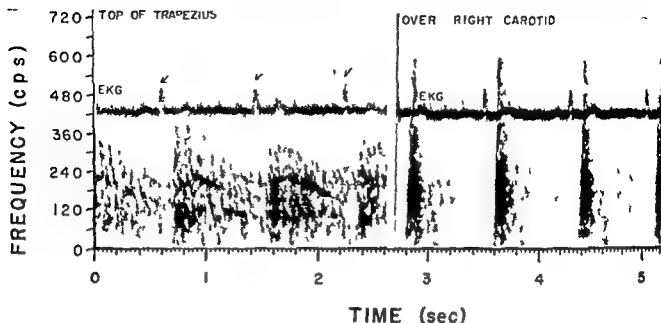


FIG. 214 Systemic pulmonary fistula

A. I. B. (702100) 31 year old male had a right upper lobe lesion previously interpreted as tuberculous. A continuous murmur with high pitched musical quality was present in the right supraclavicular fossa and could not be obliterated by pressure. B. P. was 110/60 mm Hg. X-ray of the chest showed abnormally large vessels extending from the right hilum to a lobulated lesion located at the extreme right apex behind the clavicle. The recording over the ridge of the trapezius (left) showed a continuous murmur with the general pattern seen in peripheral AV fistulae but with a rather unidirectional pattern especially as regards the harmonic occurring in diastole and located at a frequency of about 600 cps. Over the right carotid (right) there was an impet type of sound suggesting high flow in that area; the sound had the appearance of a carotid bruit (cf. 210 and 211).

At operation both the supraclavicular fossa and the thorax were explored. The first intercostal artery was several times normal size and there appeared to be communications between it (as well as small branches of the right subclavian artery) and the pulmonary vasculature in the right upper lobe. These communications were severed. After operation the murmur was no longer present.

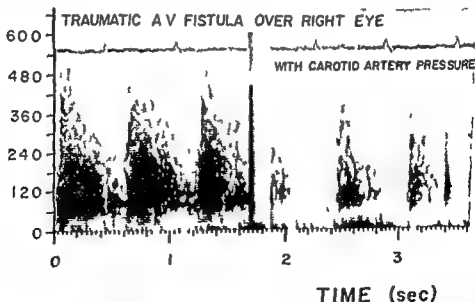


FIG. 215 Traumatic AV fistula

Recorded over right eye in I. G. (451462) 17 year old male with an AV fistula in the right frontal area following skull fracture. The intensity of the murmur is greatly reduced by pressure on the right carotid. Note that in the record on the left the continuous murmur has an abrupt marked accentuation at a distance after the QRS consistent with the time necessary for transmission of the arterial pulse from the heart and that the top of the murmur has roughly the shape of an arterial pulse pressure curve.

Brockbank suggested that the murmur is especially likely to develop if cervical ribs are present.

In coarctation of the aorta there may be a striking murmur often continuous over the vertebral collateral.

### Uterine Souffle

The uterine souffle is of arterial origin, that is, it arises in the uterine arteries which are called on to carry a much increased volume of blood because of the pregnant state of the uterus. The arterial nature of this murmur is proved by the shape of the fundamental in the case of the murmur of uterine souffle (Fig. 200) and by the fact that there is an appropriate delay after the first sound in the case of the normal uterine souffle. Burwell (202) points out the functional similarity between the placenta and an AV fistula. The identity of the uterine souffle and the murmur of AV fistula strengthens the analogy.

The blood flow to the uterus in pregnancy is about 600 cc/min on the average (202A). Relatively high pressure and oxygen content of uterine arterial blood support the view that the placenta is a low resistance area.

The uterine souffle is usually heard over one or the other side of the abdomen in the late stages of pregnancy. It is a phenomenon which is by no means constant but which tends to come and go for no apparent reason. Position of the fetus with pressure on or other distortion of the uterine arteries may be a factor. Uterine souffle is not a specific sign of pregnancy since it may occur in association with a large myomatous uterus or large ovarian tumor. The fact that the rate of the uterine souffle corresponds to the maternal heart rate facilitates differentiation from the fundal souffle and the murmur of fetal cardiac malformation; the rate in the latter two instances is that of the fetal heart.

### Mammary Souffle

In late pregnancy and during the early postpartum period one may hear over the upper margin of either breast or usually both a murmur which is full blown and continuous (Fig. 218) with little accentuation like the machinery murmur of patent ductus arteriosus. When present in less than full blown form it can be recognized by ear (and demonstrated by recording) that the mur-

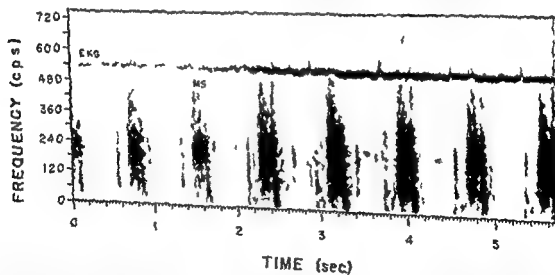


FIG. 18. Mammary souffle recorded over the upper margin of the breast early post partum in L.C. (22399). The murmur in this recording is aortic in type, isolated and extended across the second and third spaces. It was essentially complete in character during the recording when pressure was applied to the microphone there was evenling for intensity of the murmur. On release of pressure the murmur became almost continuous. Although this phenomenon occurs in full blown form in a relatively small percentage of cases we have observed at least two others (A.C. 42246 and J.C. 42247) with a striking change in tone when lying



venous blood returning from an affected limb is increased as in any AV fistula. Furthermore, not only is there increased heat over the involved areas, but also a bruit may be audible, for example, over the skull and tibia. When in the skull the murmur may be troublesomely audible to the patient. Poppen (1217) suggested that narrowing of foramina at the base of the skull with constriction of arteries might be the mechanism.

A murmur may be heard over the spleen when it is enlarged from any cause (107A). The murmur is likely to have an arterial pattern with a peak in late systole, occasionally it is continuous. A syndrome of splenic arteriovenous fistula with ascites and the characteristic continuous murmur over the lower posterior portion of the left rib cage has been described (241C). Because of the significant occurrence of aneurysm formation of the splenic artery with rupture during pregnancy, the occurrence of this syndrome in the puerperium is probably of note.

Cavernous hemangioma of the liver, a vascular hamartoma, occurs somewhat more often in the left lobe and in females (1387A). A murmur, either systolic or continuous, and sometimes with thrill may be an important clue to its presence. The lesion is frequently amenable to resection.

Partial occlusion of a peripheral artery produces a systolic murmur with the Christmas tree configuration of an ejection stenosis murmur. The peak of the murmur appears to coincide with the peak of the arterial pressure pulse. Occasionally, the murmur may be continuous in accordance with an important principle which was first elucidated by Myers and colleagues (1136) and to which I have had occasion to refer already. When the obstructive disease is sufficiently severe or collateral circulation is inadequate or pressure distal to the obstruction is lowered through vasodilatation the murmur may be continuous simply because pressure proximal to the obstruction is at all times sufficiently higher than that distal to the obstruction to produce a murmur. Myers and his colleagues showed that a systolic murmur of partial obstruction of the artery to a limb could be converted into a continuous one by having the subject exercise that limb. The interpretation was that vasodilatation instigated by exercise resulted in a drop in pressure distal to the obstruction—

the condition necessary for a continuous murmur.

The value of auscultation over peripheral vessels in cases of suspected or proved peripheral vascular disease cannot be overemphasized (1448). In atherosclerosis, narrowing of the ostium of the left subclavian with systolic murmur in the left supraclavicular fossa and reduced blood pressure in the left arm is fairly frequent (922).

In the aortic arch syndromes (1312)—narrowing or obliteration of the mouths of the great vessels arising from the arch of the aorta—a continuous murmur simulating that of patent ductus arteriosus may be heard in the vicinity of the clavicle especially in cases of almost total obstruction of all branches at the arch. The mechanism is clearly that elucidated by Myers and his colleagues (1136). A systolic murmur from lesser grade of aortic obstruction occurs rather frequently (837).

Hinohara (68a) of Kyoto devised a method for recording sounds from the esophagus. He concluded that the method has particular virtue for recording from the vicinity of the aortic arch. In a patient with the young female arteritis variety of the aortic arch syndrome (which seems to occur unusually frequently in Japan) he recorded a continuous murmur from the esophagus in the vicinity of the arch. Presumably the continuous murmur could not be detected on the surface of the chest.

Edwards and Levine (405) point out that a systolic murmur heard in the supraclavicular fossa or axilla with the arm in certain position can be a useful clue to the diagnosis of thoracic outlet syndrome. Compression of the subclavian artery is the mechanism. In the thoracic outlet syndrome arterial obstruction may be progressive because of the secondary atherosclerosis which is incited at the site of repeated trauma to the artery.

In the 1920 edition of his small book on heart disease Brockbank (182) wrote as follows (p. 97):

A very well marked systolic murmur simulating exactly an aortic murmur is sometimes heard over the substernal stern and base of the heart in quite healthy persons who are holding, the shoulders well back. It may be heard when the subject being examined is holding his vest well up for auscultation at the base of the heart. It disappears at once if the shoulders are brought forward to remove the pressure

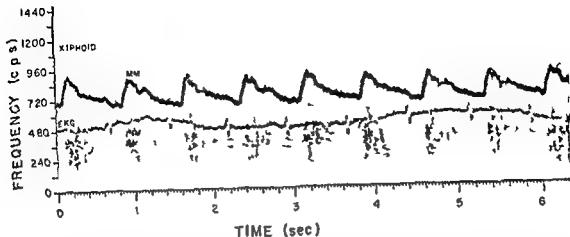


Fig 20 Musical Cruveilhier Baumgarten murmur of arterial type

In B 9 a 40 year old man with Laennec's cirrhosis a dilated tortuous vessel seemed to perforate to a pleural (anous) location in the angle between the xiphoid and the left costal margin. It coursed cephalad along the left sternal border and appeared to perforate into the anterior mediastinum at the level of the fifth intercostal space. The vessel was thus walled like a vein but had an arterial pulse. Over it was a musical murmur (MM) with the appearance shown here. It was made up of a continuous pure tone (a single harmonic or fundamental) with the shape of an arterial pulse pressure curve. It was located at a higher frequency level than that of most musical arterial murmurs (cf Figs 201 and 202). There was an intermittent noisy murmur (NM) at a lower frequency level. As in other recordings the rise and fall of the MM indicated inspiration and expiration respectively. There was no definite change in the murmur with re-piration or with the Valsalva maneuver. Localized pressure at either the caudal or the cephalad point of perforation obliterated the murmur. The murmur was audible for a distance of only about two inches from the anomalous vessel.

A more conventional venous hum had been present for at least two months before the appearance of this dilated vessel. The arterialized nature of the blood was indicated by an oxygen content of 96 per cent. Increased hepatic arterial blood flow in alcoholic cirrhosis opening of an anastomosis between hepatic arterial radicles and portal vein radicles and indirectly the development of communications between the hepatic artery and porto-caval venous collaterals were suggested. In this case blood was thought to be sluiced fairly directly from a branch of the hepatic artery to a venous collateral draining into the caval system. In essence there was an arteriovenous fistula.

It should be stated that the man had had plenectomy previously. It is my opinion that the anomalous vessel and its murmur were not related to this procedure.

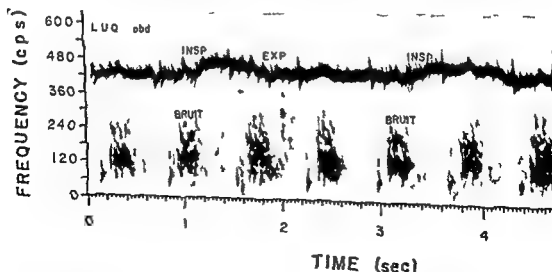


Fig 201 Hepatic bruit associated with massive liver metastases

In B (1253b) 51 year old male has massive liver metastases from an adenocarcinoma of the sigmoid colon. The murmur over the left lobe of the liver begins at about 0.12 sec after the first sound. Since it occurs with the peak of arterial flow it is probably arterial in origin.

mur begins rather abruptly in late systole, as judged by its relation to the heart sounds, and extends slightly into early diastole. The murmur is fickle in its intensity and has a rather superficial quality. Slight pressure on the stethoscope may change its quality and confer upon it partial musicality. It can be obliterated entirely by moderately firm pressure with the stethoscope (Fig 218 B) although a lesser degree of pressure may exaggerate it. I believe it is of arterial origin. It bears, therefore, some interesting similarities to uterine souffle which likewise is produced through the increased demand of an organ for blood supply resulting from augmentation of a physiologic function. The observations (533)(733) that a striking venous pattern over the breasts is demonstrable by infrared photographs in these patients and that the murmur can be obliterated by pressing with the edge of the hand at a point between the stethoscope and the main part of the breast are not inconsistent with an arterial origin of the murmur. With any hyperemia of the breast a prominent venous pattern is to be expected. Pressure distal to the stethoscope can be expected to interfere with arterial flow.

Obviously, the practical importance of the phenomenon resides in the very real risk of confusing the murmur with one of cardiac origin. In some cases

a congenital heart lesion has been suspected and special diagnostic studies performed. I like so many other situations in medicine in general and in connection with cardiovascular sound specifically, knowledge that the phenomenon may occur is the main insurance against diagnostic error.

The mammary souffle has been referred to by Gilston and McPhaul (553), Grant (584), Bonham Carter and Walker (132), Scott and Murphy (1371), and Jones (773). The last writer emphasized that it may be a musical murmur like the uterine souffle, which it resembles also in variability and mechanism. In 1949 Wells and colleagues (1529, Fig 31) described what they called a mediastinal hum in a 24 year old, seemingly normal, pregnant woman. The murmur was heard over the lower central sternum and following delivery (the murmur was less intense and rather more continuous). This may have been a mammary souffle.

### The Cruveilhier Baumgarten Murmur

The Cruveilhier Baumgarten murmur (946) is heard over the venous collaterals, connecting the portal and caval venous systems on the abdominal wall. Laennec's cirrhosis of the liver is the most frequent cause of the portal hypertension which results in development of these collaterals—

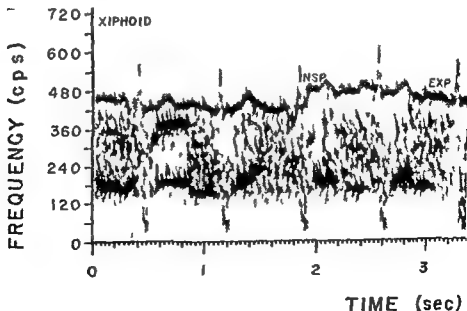


FIG 219 Cruveilhier Baumgarten murmur in V. B. (765876) 66 year old woman with primary biliary cirrhosis. The murmur was well localized to the xiphoid and often exclusively diastolic, being easily confused for an aortic diastolic murmur. In this recording it is continuous and has a musical quality.

side of the hand on the abdominal wall between the site of cultivation and the umbilicus.

Herrick (671A) Dock (360A) and others have proposed that there is a relative and probably an absolute increase in hepatic arterial blood flow in Laennec's cirrhosis; that there are abnormally wide arterioportal anastomoses constituting in effect arteriovenous fistulae and that they contribute to the portal hypertension. It is entirely possible that the murmur recorded over the liver at laparotomy is generated in these communications. It is less likely that the murmur recorded at the skin surface is of this origin. However the case shown in Figure 220 would support the notion that the murmur is generated in arteriovenous fistulae. The continuous murmur is mixed with a harmonic which describes an arterial pulse pressure curve. The murmur was well localized to the angle between the xiphoid and left costal margin where a tortuous dilated vein-like structure displayed an arterial pulsation. The murmur is thought to be the result of abnormal communications from the hepatic artery to the portal vein and eventually to these collateral venous channels according to the mechanism described by Herrick and Dock.

Melickson and Criss (1044) of Hongkong have studied a case with a murmur of the type shown in Figure 220 and provided convincing evidence that it is generated in hepato-portal venous arteriovenous fistulae which develop by the mechanism of Herrick and Dock. The murmur was continuous with a tollie accentuation. At laparotomy the murmur could be obliterated by occluding the hepatic artery. Occlusion of the portal vein had no effect on the murmur. Lipoidal arteriograms of the liver obtained at autopsy in the same case demonstrated passage of the contrast medium from the hepatic artery into large branches of the portal vein. Although they termed the murmur a venous hum in their title they concluded that it differed in many respects from that described by other and probably had its origin in arteriovenous hums in the liver.

Let no one think that the differentiation from cardiac murmurs is always obvious. Green (621) described cases in which the continuous murmur was limited to the precordial area and was taken to indicate congenital heart disease. He suggested

with some basis in anatomical observation that in many of these cases a sizable venous sinus lies immediately beneath the lower sternum. Subcutaneous collateral often penetrates in the vicinity of the xiphoid joint. Localized pressure with the finger tips at the point of penetration may obliterate the murmur.

### Cephalic Bruits

As a strange human fault, no cultivation of the skull seems to be the one thing most likely to be neglected in a routine neurological examination. Cephalic auscultation is a forgotten practice and even when the patient calls attention to the fact that he hears noises in his head they are so likely to be ascribed to some form of auditory hallucination or to tinnitus that the examiner rarely thinks of checking the patient's statement with a stethoscope.

Cullen and Bailey (3925) (321A)

Cephalic bruits (1009) (1149) have long been recognized (see Fisher on p. 17) but recently have enjoyed a well-deserved revival of interest. They are a heterogeneous class comprising murmurs generated in arteriovenous fistulae (Fig. 223) some of which are probably venous hums produced in the large and others generated in arteries narrowed for one reason or another. Ocular bruits (277) murmurs heard over the orbit are a variety. Cephalic bruits are among the dramatic aspects of medicine in this respect rivaling the very loud heart murmur especially that of retroverted aortic cusp (p. 269). The interesting anecdotes concerning noises heard by the patient himself diagnosed made over the telephone (627) etc. are similarly legion in these two types of case. Ocular bruits are detected by applying the bell of the stethoscope to the upper lid of the closed eye. Cephalic bruits are often especially disturbing to patients because of the peculiar noise. The complaint of roaring in the head or of the hearing of any other variety of unusual noise should prompt cultivation of the cranium including the eyeball before the symptom is interpreted as tinnitus or worse psychoneurosis.

Disappearance with carotid occlusion is a helpful indicator that the sound is of a vascular origin and generated above the neck (1217). It is possible of course for the murmur to continue because the vertebral artery has not been occluded.

the Crueveilhier Baumgarten syndrome (28) (The Crueveilhier Baumgarten disease (236, 657) is a specific congenital malformation in which the umbilical vein remains open after birth and cirrhosis of the liver develops secondary to the short circuiting of blood away from the liver) The murmur is essentially a venous hum (673, 1018) It is best heard in the epigastrium and less well heard below the umbilicus and over the lower thorax It occasionally is limited to the xiphoid or lower sternum For this reason and the fact that it may be predominantly or exclusively diastolic in aortic diastolic murmur may be simulated It is frequently musical in quality (520), and analogies to a humming, bee come immediately to mind (Fig. 219) There may be accompanying thrill There may be striking respiratory variation with accentuation in inspiration Two factors may favor inspiratory accentuation increase in intra abdominal pressure, decrease in intrathoracic pressure Occasionally the murmur is constant in intensity resembling the sound produced by a sea shell held against the ear (117) In such cases it is less likely to be influenced by respiratory cycle or posture The Crueveilhier Baumgarten murmur is usually maximal when much ascites is present and likely to disappear after abdominal par-

centesis It is often loudest with the patient in the upright position

In cases in which the Crueveilhier Baumgarten murmur is not audible on auscultation of the abdomen it may be heard (1064) on direct auscultation of the liver at laparotomy In such cases, the murmur tends to become louder as one approaches the porta hepatis

Although the Crueveilhier Baumgarten murmur can legitimately be considered a venous hum the accentuation is frequently in ventricular systole not diastole as in the case of venous hums heard in the neck The fact that it may have systolic accentuation may make it difficult to distinguish from the continuous murmur of a hemangioma of the liver, an intra abdominal AV fistula or a vascular tumor such as a chorioepithelioma Furthermore a very large liver which is the site of multiple metastase of a neoplasm (Figs. 221 and 222) or is involved by primary carcinoma of the liver may produce enough distortion of and pressure on, the hepatic artery that a continuous murmur with systolic accentuation occurs Before an epigastric bruit can be identified as a Crueveilhier Baumgarten murmur there should be evidences of dilated subcutaneous veins A useful clue to the demonstration of obliteration of the hum by pressure with the

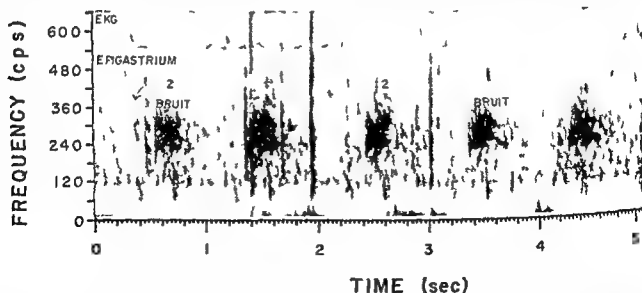


FIG. 222 Arterial souffle

Recorded in epigastric area of 34 year old male (G F 557871) with Hodgkins disease for which a great deal of x ray therapy had been given to the upper abdomen The spleen was little enlarged The murmur was thought to be produced in an artery compressed by post radiation scarring The sound is concentrated at a frequency of about 300 cps and has the appearance of the top cut off an arterial pulse pressure curve Background noise

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Cushing and Buley 1924 (321A)

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the Cruevillier Baumgarten syndrome (28) (The Cruevillier Baumgarten disease (256, 657) is a specific congenital malformation in which the umbilical vein remains open after birth and cirrhosis of the liver develops secondary to the short circuiting of blood away from the liver) The murmur is essentially a venous hum (673, 1018) It is best heard in the epigastrium and less well heard below the umbilicus and over the lower thorax It occasionally is limited to the epiphoid or lower sternum For this reason and the fact that it may be predominantly or exclusively diastolic, in aortic diastolic murmur may be simulated It is frequently musical in quality (520), and analogies to a humming bee come immediately to mind (Fig. 219) There may be accompanying thrill There may be striking respiratory variation with accentuation in inspiration Two factors may favor inspiratory accentuation: increase in intra abdominal pressure, decrease in intrathoracic pressure Occasionally, the murmur is constant in intensity resembling the sound produced by a set held against the ear (117) In such cases it is less likely to be influenced by respiratory cycle or posture The Cruevillier Baumgarten murmur is usually maximal when much ascites is present and likely to disappear after abdominal para-

centesis It is often loudest with the patient in the upright position

In cases in which the Cruevillier Baumgarten murmur is not audible on auscultation of the abdomen it may be heard (1064) on direct auscultation of the liver at laparotomy In such cases, the murmur tends to become louder as one approaches the porta hepatis

Although the Cruevillier Baumgarten murmur can legitimately be considered a venous hum, the accentuation is frequently in ventricular systole not diastole as in the case of venous hums heard in the neck The fact that it may have systolic accentuation may make it difficult to distinguish from the continuous murmur of a hemangioma of the liver, an intra abdominal AV fistula or a vascular tumor such as a chorioepithelioma Furthermore, a very large liver which is the site of multiple metastases of a neoplasm (Figs. 221 and 222) or is involved by primary carcinoma of the liver may produce enough distortion of and pressure on the hepatic artery that a continuous murmur with systolic accentuation occurs Before an epigastric bruit can be identified as a Cruevillier Baumgarten murmur, there should be evidences of dilated subcutaneous veins A useful clincher is the demonstration of obliteration of the hum by pressure with the

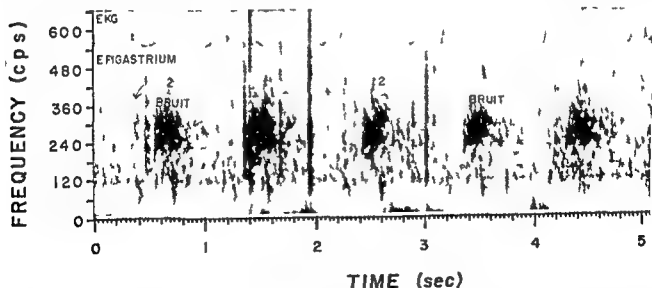


FIG. 222 Arterial souffle

Recorded in epigastric area of 31 year old male (G 1 57571) with Hodgkins disease for which a great deal of x ray therapy had been given to the upper abdomen The spleen was little enlarged The murmur was thought to be produced in an artery compressed by post radiation scarring The sound is concentrated at a frequency of about 300 cps and has the appearance of the top cut off an arterial pulse pressure curve Background noise

side of the hand on the abdominal wall between the site of auscultation and the umbilicus.

Herrick (6744) Dock (3604) and others have proposed that there is a relative and probably in absolute increase in hepatic arterial blood flow in Laennec cirrhosis that there are abnormally wide arteriportal anastomoses constituting in effect arteriovenous fistulae and that the contribution to the portal hypertension. It is entirely possible that the murmur recorded over the liver at laparotomy is generated in these communications. It is less likely that the murmur recorded at the surface is of this origin. However the case shown in Figure 220 would support the notion that the murmur is generated in arteriovenous fistulae. The continuous murmur is mixed with a harmonic which describes an arterial pulse pressure curve. The murmur was well localized to the angle between the xiphoid and left costal margin where a tortuous dilated vein like structure displayed an arterial pulsation. The murmur is thought to be the result of abnormal communication from the hepatic artery to the portal vein and eventually to these collateral venous channels according to the mechanism discussed by Herrick and Dock.

McFadden and Cray (1061) of Hongkong have studied a case with a murmur of the type shown in Figure 220 and provided convincing evidence that it is generated in hepato-portal venous arteriovenous fistulae which develop by the mechanism of Herrick and Dock. The murmur was continuous with systolic accentuation. At laparotomy the murmur could be obliterated by occluding the hepatic artery. Occlusion of the portal vein had no effect on the murmur. Ipsoidal arteriogram of the liver obtained at autopsy in the same case demonstrated passage of the contrast medium from the hepatic artery into large branches of the portal vein. Although they termed the murmur a venous hum in their title they concluded that it differed in many respects from that described [by others] and probably had its origin in arteriovenous hums in the liver.

Let no one think that the differentiation from cardiac murmurs is always obvious. Cray (621) described cases in which the continuous murmur was limited to the precordial area and was taken to indicate congenital heart disease. He suggested

with one brain in anatomical observation that in many of these cases a sizable venous mass lies immediately beneath the lower sternum. Subcutaneous collateral often penetrate in the vicinity of the xiphoid-sternal joint. Localized pressure with the finger tip at the point of penetration may obliterate the murmur.

### Cephalic Bruits

By a strange human frailty auscultation of the skull seems to be the one thing most likely to be neglected in a routine neurological examination. Cephalic auscultation is a forgotten practice and even when the patient calls attention to the fact that he hears noises in his head they are so likely to be ascribed to some form of auditory hallucination or to tinnitus that the examiner rarely thinks of checking the patient's statement with a tetho cope.

Cushing and Bailly (1925) (3213)

Cephalic bruit (1009) (1169) have long been recognized (see Fisher on p. 17) but recently have enjoyed a well-deserved revival of interest. They are a heterogeneous class comprising murmur generated in arteriovenous fistula (Fig. 223) some of which are probably venous hums produced in the large sinuses others generated in arteries narrowed for one reason or another. Ocular bruits (277) murmurs heard over the orbit are a subcategory. Cephalic bruit are among the dramatic aspects of medicine in this respect rivaling the very loud heart murmurs, especially that of retroverted aortic cusp (p. 209). The interesting anecdote concerning noises heard by the patient himself diagnoses made over the telephone (627) etc etc is hardly legion in these two types of case. Ocular bruit are detected by applying the bell of the tetho cope to the upper lid of the closed eye. Cephalic bruit are often especially disturbing to patients because of the peculiar noise. The complaint of roaring in the head or of the hearing of any other variety of unusual noise should prompt auscultation of the cranium including the occiput before the symptoms interpreted as tinnitus or worse psychoneurosis.

Disappearance with external occlusion is a helpful indicator that the sound is of vascular origin and generated above the neck (1217). It is possible of course for the murmur to continue because the vertebral artery has not been occluded.



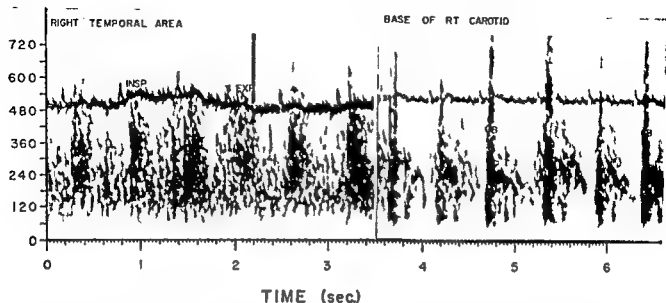


FIG. 223 Cephalic bruit recorded in the right temporal area in V. M. (B18376) 18 month old child. The continuous murmur has a slight systolic accentuation characteristic of arteriovenous fistula or at least indicative of arterial origin. An origin in the venous sinus, for example, is unlikely. A congenital vascular malformation appears to be present. Carotid bruit and probable muscular venous hum in the same patient. Recorded at the base of the neck on the right. About 0.12 sec. after the QRS there is a rather long transient. It is followed by a single harmonic and there is an inconstant harmonic in diastole. The second sound is in a normal position. The transient is probably produced at the bifurcation of the innominate. The musical elements may be of venous origin. With arteriovenous fistula in the head, carotid bruit and venous hum are more likely to occur.

An interesting phenomenon is accentuation of the bruit of an intracranial lesion when pressure is applied to the *contralateral* carotid artery. Apparently the maneuver results in increased flow through the area where the murmur is generated. The possibility of a transmitted cardiac murmur must always be kept in mind and excluded by precordial auscultation. The neurologist must keep other systemic disease in mind in this as in other situations and must remember that fever or anemia may be responsible for cephalic bruits. In our present state of knowledge cephalic bruits in children are always of more doubtful significance than those in adults. Poppen (1217) states that a bruit is very frequently present over the mastoid process of children. He thought it to be generated at the point where a vessel penetrates the skull and claimed it could be obliterated by local pressure. In hemiplegic patients or patients with neurologic manifestations which may be on a vascular basis, auscultation of the head is indicated although the clinician must guard against being confused thereby and uncovering more 'red herrings' than worthwhile information. He must be prepared to be unable to interpret the findings.

In Hamburger's classical paper (627) one patient, Mrs. McC., still had an unexplained cephalic bruit ten years after onset and no more diagnostic features or adverse events had developed. In another patient, a Baltimore physician who died only recently at the age of 84 years, the murmur disappeared spontaneously a few months after it was first heard and some 30 years before his death.

Mackenzie (1010) described 11 patients with cephalic bruits. The responsible lesions included cerebral angioma, meningioma, carotid aneurysms (both congenital and traumatic), tumor of the glomus jugulare, incipient internal carotid occlusion, pinealoma, and cerebral thrombophlebitis.

The classical example of intracranial arteriovenous fistula with murmur is that between the carotid artery and the cavernous sinus (1302). There is likely to be pulsating exophthalmos and the same continuous murmur which characterizes other arteriovenous fistulas. Trauma is the cause in about 70 per cent of cases although in inherent vessel weakness may contribute. When exophthalmos is bilateral (as occurs in about 10 per

vert of eyes) the side on which the murmur is louder is usually the side of the fistula. The murmur may have it on either side before the appearance of exophthalmos or congestion of the fundal vessel (1501). The loudest murmur we have heard in carotid-cavernous fistula was such a one (G T 681311). It is postulated that the communication is small in such case with small hunt and little effect on cavernous sinus pressure but with generation of a loud murmur.

Arteriovenous fistulas may follow skull fracture just as they may occur in the thoracic wall following rib fracture. In extreme anemia in patients with arterio-sclerosis there may be marked murmur. Probably the arterio-sclerosis need not be severe. A physician described (23) who had two gastro-intestinal hemorrhages with each of which a noise in the head was associated. Cohen and Miller (277) proved the presence of arterio-sclerotic narrowing of the carotid siphon in two patients with eyeball bruit and strongly suspected it in a third. There was hemangioma in one case and profound anemia in two. A venous thrombosis of the internal carotid artery. A significant point is that in six of the seven patients reported by Cohen and Miller (277) the bruit was limited to the eyeball. In applying the ophthalmoscope to the lid over the closed eye the patient should try to relax the extra-ocular muscles as much as possible to minimize sound conceal a vascular bruit or be mistaken for it. If the physician holds down the lid he is listening over and lets the patient keep the other eye open muscle sound may be avoided. In children a cephalic bruit may occur merely with increased intracranial pressure. Non vascular brain tumors may be accompanied by cephalic bruit sometimes perhaps through direct pressure of the tumor on vessel more often through influence of the hydrocephalus.

In one infant a physiologic fontanel bruit is heard over the fontanel (582). Although one has thought the fontanel bruit to be of arterial origin it seems more likely that it arises in the vein. After closure of the fontanel the bruit disappears in essentially all normal individuals. Dalgaard Nielsen (326) after surveying about 3000 individual provided the following figures on incidence

|            |     |
|------------|-----|
| 1 yr       | 3%  |
| 1-2 yr     | 17% |
| 1-1 1/2 yr | 19% |
| 1 1/2-2 yr | 7%  |
| 2-2 1/2 yr | 2%  |
| 2 1/2 yr   | 0%  |
| Adults     | 0%  |

Henoch (672) thought the fontanel bruit to be a specific sign of ricket. Delayed closure of the fontanel was the probable basis of the association.

Wadli and Monckton (1498A) have provided an extensive study of intracranial bruit. In a group of 228 adults who seemed to be normal they found a bruit in three. An evoked bruit that is one heard with contralateral carotid artery compression may persist into middle life. A familial aggregation of cases of intracranial bruit in normal person is observed. The authors postulated a gene determined variation in the pattern of the circle of Willis. Anemia and thyrotoxicosis may bring out a tendency to intracranial bruit. The pathologic lesions for bruit included cerebral angioma, orbital angioma, carotid-cavernous fistula, Piget disease of the chiasm, tumor (especially meningioma and glomus jugularis tumor) and arterio-sclerosis of the internal carotid artery.

One should listen for an intracranial bruit especially in cases of migraine, epilepsy or subarachnoid hemorrhage. Intracranial angioma or arteriovenous malformation may present in any one of these forms.

#### FUNCTIONAL HEART MURMURS

Men are as often deceived by their ears as by their eyes and they may hear ghosts as well as see them.  
Latham 1547

Full of sound and fury, signifying nothing.  
Shakespeare *Macbeth*

Functional murmurs represent the most difficult subject in the entire domain of cardiovascular sound (456-974-985A). Various names have been suggested for this category of murmur: accidental, unexplained benign innocent cardiopulmonary, not significant, humic etc. Some of these terms obviously have certain special significance and none of them is entirely satisfactory. In a sense all murmurs are functional and humic. The murmur of atrial septal defect (see p. 344) has

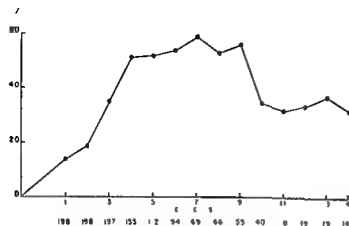


FIG. 224 Age incidence of precordial systolic murmurs in normal infants and children (total of 1264 patient years.) (Courtesy of Epstein (431) and *Journal of Pediatrics*.)

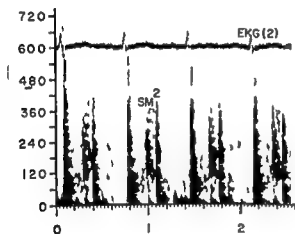


FIG. 225 Extracardiac murmur

Circumscribed late systolic murmur in patient A H (A81746) 11 years old with history of rheumatic fever two years previously. A persistent apical systolic murmur had been occasion for concern and had been interpreted as indicative of mitral regurgitation. Because of its circumscribed character and abrupt onset extracardiac origin is likely.

its auscultatory features and mechanism in common with some functional murmurs yet cannot be considered innocent. Some of the murmurs which accompany rheumatic carditis, severe chronic anemia, vitamin deficiency, heart failure dilatation of the heart thyrotoxicosis, and so on, are also functional, but by no means innocent. Review of the literature leads to the conclusion that all loud systolic murmurs and all diastolic murmurs regardless of intensity are usually considered 'organic', all systolic murmurs of low intensity are usually considered functional.

The problem is emphasized by the fact that in 100 per cent of children one can record (1033) at least some slight systolic murmur. Furthermore with sufficiently sensitive pick ups one can record a systolic murmur in all normal 20 year old subjects. Finally, intracardiac phonocardiography and direct phonocardiography reveal a systolic murmur in or over the pulmonary artery in all cases. With stethoscopy alone, the incidence of a systolic murmur is very high in certain age groups (Fig. 224).

In the following discussion "functional" and "innocent" will be considered synonymous. What murmurs can be identified as 'functional' with fair certainty?

1. Circumscribed systolic murmurs such as (a) the late systolic variety (see Figs. 225 to 229), especially if introduced by a systolic click (208), and (b) the mid systolic murmur which is separated from both the first and the second sound by a brief gap (Fig. 230 and 231 also Fig. 20a) are quite clearly of extracardiac origin, probably from roughening of the pericardium, and are therefore innocent. The circumscribed mid systolic murmur may have a grating quality (208) consistent with its origin in pericardial roughening. It sometimes persists for several weeks or months or possibly indefinitely after acute pericarditis. (Occasionally the murmur of aortic stenosis may show a gap between the first sound and its onset, and of course the usual gap after the murmur. Possibly the presence of bundle branch blocks increases the likelihood of a gap preceding the murmur of aortic stenosis.)

The late systolic murmur introduced by a systolic click illustrated by Bridgen and Leithim (177) is probably an example of a pericardial murmur rather than of mitral regurgitation. The fact that both may occur after rheumatic fever may cause confusion.

The late systolic murmur has long plagued clinicians (296) and cannot honestly be said to be completely understood at present (704). In 1903 Hall (62a) mentioned the view that it is due to mitral regurgitation with the valve competent only in the first part of systole, an unlikely view. Evans (441) emphasized its innocence. The murmur under discussion occurs at the apex or left midprecordium. If phonocardiograms show that

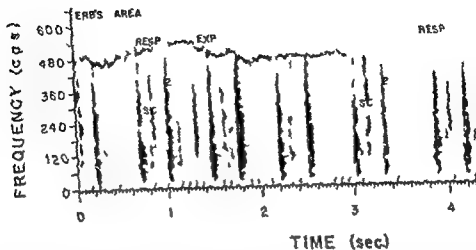


Fig 26. Late systolic murmur and systolic clicks

D C (48601) 23 year old female was sent for exam because of an (initially discovered) murmur. The murmur was present at the left (third) border only with the patient in the recumbent position. There was no previous history that seemed pertinent. The character of the murmur (apex not known) and the timing (introduced) by a click—is unusable evidence of no late systolic clicking from this area. Followed only the click.

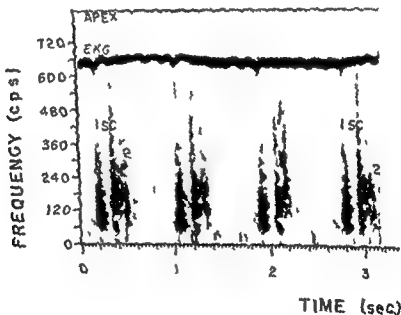


Fig 27. Extra-systolic murmur and clicks

L H (74164) 34 year old male was rejected for armed service 13 years before because of a murmur. His history is active in athletics and in his work is a farmer. Detailed investigation including right heart catheterization reveals no abnormalities. Apex in left lateral decubitus. The first late systolic clicks now introduced a late murmur. The recording was made at the apex in the supine position. There were multiple late systolic clicks.

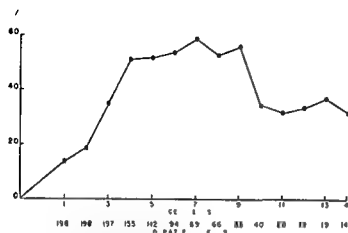


FIG. 221 Age incidence of precordial systolic murmurs in normal infants and children (total of 1761 patient years) (Courtesy of Epstein (131) and *Journal of Pediatrics*)

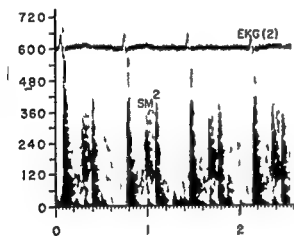


FIG. 222 Intracardiac murmur

Circumscribed late systolic murmur in patient A II (AS1746) 11 years old with history of rheumatic fever two years previously. A persistent apical systolic murmur had been occasion for concern and had been interpreted as indicative of mitral regurgitation. Because of its circumscribed character and abrupt onset extracardiac origin is likely.

its auscultatory features and mechanism in common with some functional murmurs yet cannot be considered innocent. Some of the murmurs which accompany rheumatic carditis, severe chronic anemia, vitamin deficiency, heart failure, dilatation of the heart, thyrotoxicosis and so on, are also functional but by no means innocent. Review of the literature leads to the conclusion that all loud systolic murmurs and all diastolic murmurs regardless of intensity are usually considered "organic", all systolic murmurs of low intensity are usually considered "functional".

The problem is emphasized by the fact that in 100 per cent of children one can record (1033) at least one slight systolic murmur. Furthermore with sufficiently sensitive pick ups one can record a systolic murmur in all normal 20 year old subjects. Initially, intracardiac phonocardiography and direct phonocardiography reveal a systolic murmur in or over the pulmonary artery in all cases. With stethoscopy alone, the incidence of a systolic murmur is very high in certain age groups (Fig. 221).

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## MURMURS

early systole; indeed free of murmur then the late systolic murmur can probably be considered innocent.

Ci tex (13a) quotes his master Vaquez in the opinion that apical systolic rousures are organic if holosystolic non-crescendo or decrescendo (Vaquez did think that ventricular dilatation can result in a protosystolic or telesystolic murmur) Ci tex emphasizes the fact that circumcribed systolic murmurs occur even early following myocardial infarction. In fact he goes

farther to propose that such murmurs in mid or late systole occur with local infarctions but when in early systole indicate apical infarction!

2. Murmurs with striking respiratory variation in a subject with a chest deformity which makes likely compression of lung tissue by the heart are probably cardiopulmonary (see Figs. 206 and 207) and therefore innocent. Mather and Stone (1914) found a systolic murmur in essentially all cases of

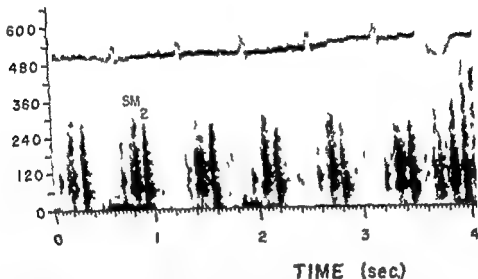


FIG. 20 Extracardiac murmur

Circumcribed mitral systolic murmur at the apex after acute pericarditis in a girl of 14 (hyalazine log 1131) in M. McD. (115-16). After persisting several months it disappeared completely. It has a rather creaking quality which is suggested by the tendency to harmonic organization.

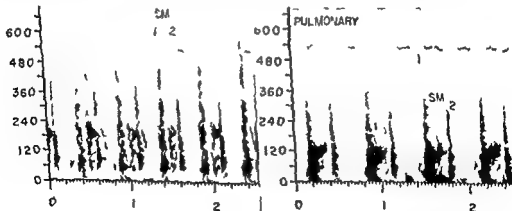


FIG. 21

FIG. 21'

FIG. 21 An extracardiac origin of the mitral murmur in an 14 year old female patient with a history of rheumatic fever; suggestive of the fact that it does not begin immediately with  $S_1$  and persists runs here & there some extent of the second  $S_1$ .

FIG. 21' Mitral systolic murmur in 14 year old patient with history of acute rheumatic fever. The mitral murmur has the spectrographic pattern of the Still murmur.

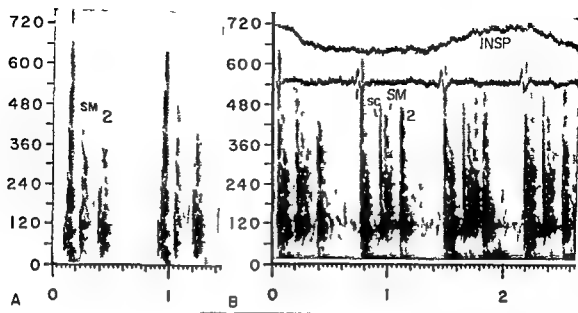


FIG. 228 Mid-systolic murmur introduced by systolic click.

1 Apex in patient (W B 674663) with Laennec's cirrhosis. No abnormality of heart and surrounding structures at autopsy. B IISB in patient (J B 149092) with chest deformity (mainly kyphosis due to a teogenic imperfection).

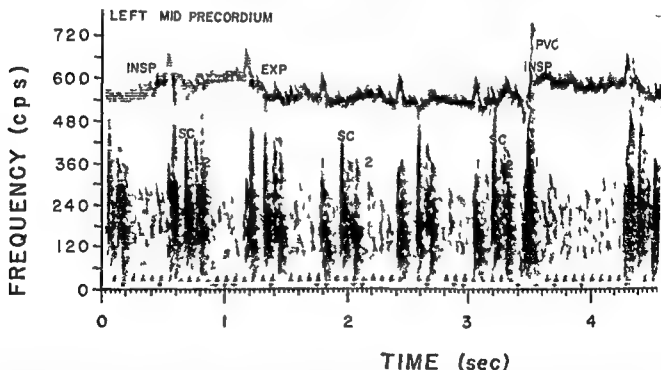


FIG. 229 Syndrome of precordial pain palpitation (cardiac neurosis and extracardiac sounds).

This recording was made from the left midprecordium of W P (768996) a 38 year old white man who had had palpitations with a sensation of skipped beats for 10 to 15 years. The palpitations always occurred when he was at rest but during period of excessive fatigue or anxiety. During the same period (and under the same circumstances) he had vague evanescent pains in the region of the left anterior axillary line. Fifteen years previously he had been rejected for armed service presumably because of his heart but was given no further details. His physician became much concerned about his condition when he heard a murmur which was however variable and placed him on digitalis. As seen in the recording the murmur is circumscribed and introduced by a click—features characteristic of extracardiac origin. One extrasystole is present (There is considerable continuous background noise either ambient or electronic between 120 and 240 cps.) The T wave changes present in the electrocardiogram may be caused by digitalis or represent residual of pericarditis.

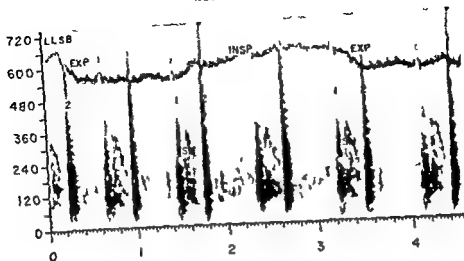


Fig. 244 Functional aortic murmur

ILSB in C. H. (20%) 16 years of male who perfectly well but has a systolic murmur first heard at the age of 9 years. The fact that the murmur is separate from both  $S_1$  and  $S_2$  may be significant as indicating its benign nature. AILSB is a relatively unusual feature characteristic of the still murmur.

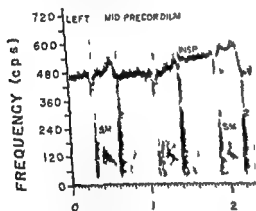
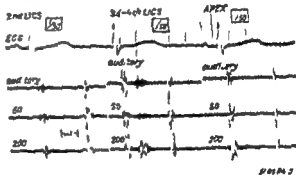


FIG 24B Functional murmur

Left mitral precut lum in "year of life" (J I 134 at)  
with apparently normal circulation



The 230°C loss may be in a still greater degree of functional manner with type. The maximum is maximum at 1150°C, paraffin from both  $S_1$  and  $S_2$  are the least a indicate that the regularity of oxidation. The three frequency channels are exactly and two other with maximum at  $\delta = 0$  at about  $\delta = 0.1$  and  $\delta = 0.2$  respectively. (From Paulin and Mannheimer (1955))

(CN) found with filtered recording that functional murmurs often had only relatively high frequency components whereas organic murmur have both high and low components (Van (1939) had predicted as follows: A detection of the frequency of the epirate murmur may also contribute to a quicker recognition of the innocent ones from among the others. This is essentially what the program does (see Figs 232 to 234) and much less satisfactorily the oscillogram. Well (1922) who designated this murmur as the coarse variety of preconal murmur noted the regular mechanical characteristic of the vibra-

tion in the scillogram and pointed out that the murmur may be very loud leading to a misdiagnosis of ventricular septal defect. Lurie (1460) wrote that a child with a normal heart may have an extraordinarily loud functional murmur which is transmitted to the back. Fogel (1467) stated that Still's murmuring, fringing murmur is the most frequent variety of functional murmur is rarely as intense as grade IV occurs most often in the years two to four. Paulin and Mannheim (1490) found that 43 per cent of functional murmurs were of this type.



funnel chest. The murmur in some cases of chest deformity may be comparable to that produced in animals by compressing the pulmonary artery. Often the murmur in milder cases of funnel chest can be eliminated by deeply held inspiration. Presented in Figure 469 is a recording of a loud pulmonary systolic murmur produced by compression of the pulmonary artery by mediastinal lympho-recoma; the murmur could be eliminated by deep inspiration. This is another instance of a functional murmur which was not innocent since it was caused by a grave condition.

3. A venous hum (p. 226), although loudest in the neck, may be audible over the upper precordium. The fact that it is loudest in the neck and can be traced continuously from the neck to the precordium aids in its identification.

4. Carotid bruit (p. 227) may be heard at the base of the neck on the right in children and may suggest mild aortic stenosis. The absence of the murmur in the aortic area assists in the correct identification.

5. Murmur souffle (p. 233) in pregnancy or the parturient state is easily differentiated if the clinician is familiar with the phenomenon.

6. Still (1446) described, and others notably and most recently Harris and his collaborators (640, 641, and 642) have studied the variety of systolic murmur which appears to be innocent and which is named for its resemblance to the twanging of a tight string. The twanging string murmur<sup>8</sup> is indeed musical by spectrographic analysis, suggesting that some elastic structure is thrown into vibration in systole. Trigonoidation of the pulmonary cusps in systole, an entirely reasonable basis for a systolic murmur (262), results in three relatively flat flaps the pulmonary cusps. It may be these that are excited to vibration. A pericardial origin is also possible. Ortiz (1167) has demonstrated the sound-producing properties of normal rubbing, serous and drawn in analogy to the group of stringed musical instruments. Milk spots roughened and thickened patches on the visceral pericardium have been suggested as sources of murmurs as far back as Gerhardt (541) in 1871. The musical character of this murmur is consistent with either

trigonoidation or pericardial rubbing. The murmur is referred to as "vibratory" by Harris (642), who points out its buzzing quality. "Groning" is the adjective, perhaps even more appropriate, suggested by Lynxwiler and Donahoe (998) and found by them to apply to 500 of 620 basal functional systolic murmurs and 120 of 620 apical functional systolic murmurs. Stuckey (1451) stated that 40 of 145 innocent murmurs were squeaky or musical. Among 300 children with systolic murmurs judged to be functional, Meseloff (1096) found that the murmur was musical in 50 per cent and in a further 6 per cent was blowing and musical. The type of murmur under discussion, although possibly not representing a homogeneous group, is probably the most frequent variety of functional murmur.

Harris and his co-workers (642) and Puhlin and Munheimer (1190) published oscillograms which suggest that Still's murmur is musical—its vibrations are regularly spaced—and does not begin quite with  $S_1$  in many instances. More recently Harris' group (643) has further confirmed the musicality by spectrographic study. Dunn (381, 387) arrived at similar conclusions from approximate estimates of frequency: that there is a large group of functional murmurs characterized by restricted frequency, namely tendency to musicality and a tendency not to begin immediately with  $S_1$  or extend completely to  $S_2$ . Bass *et al*

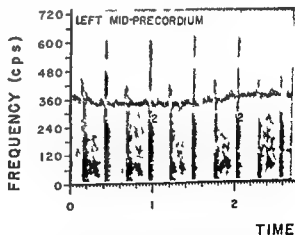


FIG. 233 Groning functional murmur

1. M. (B27352) 8 year old white male had a grade II musical groning murmur best heard at the left sternal border and varying with exercise and position. The recording (from the left midprecordium) shows the characteristic musical quality.

<sup>8</sup> Wedum and Rhodes (1518) called this the "fiddle string" murmur.

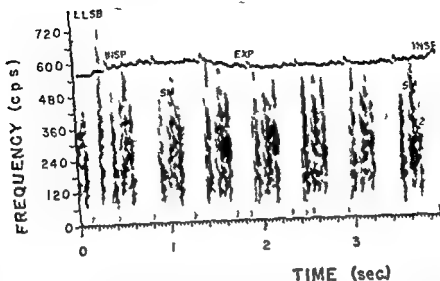


FIG. 23. Semi musical extracardiac murmur

R.P. (7/26/59) year old male was seen for murmur (strongly apparently unrelated to the heart). There was mild pectus excavatum and at the left sternal border there was a high, somewhat musical systolic murmur loudest in inspiration and in the sitting position. S1 (G (LLSB sitting)) Mitral and tricuspid closure sound seen with unusual clarity. Gap before beginning of murmur, musical element to murmur.

7. The musical late systolic murmur which varies with respiration and may be introduced by a systolic click (p. 207) is extracardiac in origin and benign in prognostic significance. The case illustrated in Figure 237 is one such although the murmur is only partially musical.

In young people with no anemia and with no evidence of dilatation of the pulmonary artery a murmur may be audible at the second, third and fourth interspace at the left sternal margin. The murmur is usually confined to the very first part of systole when the velocity of systolic ejection is maximal. It is exaggerated by exercise and the administration of adrenaline and amyl nitrate. Rapid flow through the outflow tract of the right ventricle which at this site is relatively superficial is probably the main mechanism but triangulation of the pulmonary cup is in addition an attractive possibility as a contributing factor. Usually the murmur is loudest in the supine position (1096) and after exercise (889, 1190) conditions which increase venous return and the stroke volume of the right ventricle. The lack of exercise and position by no means distinctive into the murmur of valvular lesions may have the same change. At least a faint murmur of this

type may be heard when flow is most rapid and can be recorded at the left sternal margin in the majority of young people especially after exercise and in the recumbent position and of course especially with a sensitive microphone (610). It is of note that in all subjects which a murmur can be recorded from the pulmonary artery by intracardiac phonocardiography or by direct phono-cardiography (see p. 84). The murmur heard in many instances merely the normal murmur in somewhat exaggerated form. A. Haver (1169) and ascribing the idea to Broadbent. It is not the frequency of late systolic murmurs at the pulmonary orifice that is remarkable. What is remarkable is that they are not always present. The fact that a murmur cannot be recorded directly from the inside or the outside of the heart peaks against the view that innocent systolic murmurs of aortic origin are at least a common innocent pulmonary systolic murmur in normal healthy children (1151).

Difficult to differentiate from the type of murmur which was discussed in the last paragraph and which probably can not legitimately be termed functional are the following: (1) small atrial septal defect (2) bicuspid aortic valve

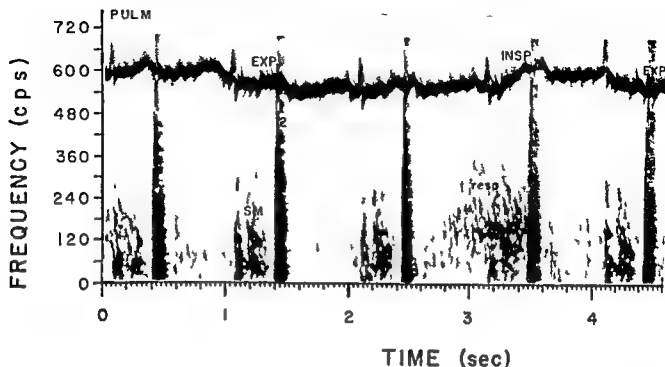


FIG. 235 Functional systolic murmur

Pulmonary area in P.M. (613214) 16 year old female who had had nosebleeds and aching of the legs from the age of 5½ years. Because of a murmur the patient was referred with the diagnosis of rheumatic heart disease. A systolic murmur at the left sternal border is rather growing in quality. The systolic murmur with a suggestion of musicality is separated slightly from both  $S_1$  and  $S_2$ .

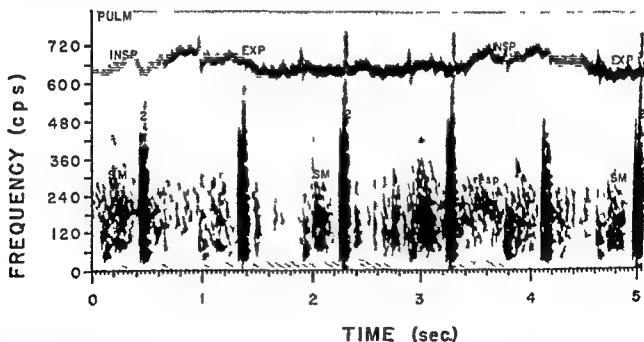


FIG. 236 Functional systolic murmur

Pulmonary area in J.M. (353828) 17 year old female followed for five years for presumed rheumatic heart disease the diagnosis being based almost exclusively on the presence of a systolic murmur of growing quality. The partially musical systolic murmur does not begin immediately with  $S_1$  and is separated from  $S_1$  by a brief gap. A third heart sound is present.



TABLE 11

## Murmurs a synthesis

|                         | Main Area                            | Radiation                        | Characteristics       |  |                           | How Best Heard   | Associated Changes in Heart Sound  | Associated Physical Signs   |
|-------------------------|--------------------------------------|----------------------------------|-----------------------|--|---------------------------|--|--|---|
|                         |                                      |                                  | Intensity (the grade) | Timing   | Pitch                     |  |  |   |
| Mitral stenosis         | Inside apex well localized           | Radiation all over to aortic     | ± to 4                | Rumble may begin with S <sub>1</sub> presystolic | Low crescendo presystolic | Left lateral decubitus after exercise bell lightly applied | All snapping opening snap increased and presystolic (usually 12 + OS)        | Diastolic especially presystolic thrill (rumble) S <sub>2</sub> murmur                      |
| Mitral regurgitation    | Apex wide area                       | Apex left back                   | 1+ to 5               | Holosystolic                                     | Intermediate              | No special technique necessary                             | All dull protodiastolic gallop P <sub>2</sub> less regular early accentuated | If with stenosis rumble louder than otherwise the case                                      |
| Aortic stenosis         | Noisy in aortic mitral area in 1 rib | Right base of neck to apex       | 1+ to 6               | Chastantree systolic                             | Wide band                 | Sitting leaning forward full expiration                    | Dull or absent A <sub>2</sub> (not invariable however)                       | Systolic thrill   |
| Aortic regurgitation    | 1 rib                                | Radiation                        | ± to 6                | Decrescendo early diastolic                      | High                      | Same as A <sub>5</sub> diastasis murmurs sometimes best    | A <sub>2</sub> may be increased normal or decreased                          | Occasional diastolic thrill Austin Flint aortic systolic murmur                             |
| Tricuspid stenosis      | Left lower sternil border            | To left mid precordial and apex  | ± to 3                | As in mitral (see above)                         | As with mitral stenosis   | Best recumbent in expiration                               | Tricuspid opening snap   | Diastolic thrill front venous waves large RA high V P presystolic pulsations of large liver |
| Tricuspid regurgitation | Right lower sternil border           | To right wall and posterior area | To 4 or 5             | As in mitral regurgitation                       | Intermediate              | No special technique necessary                             | Protodiastolic gallop  | Systolic pulse of liver veins   |

## CHAPTER 11

# Miscellaneous Varieties of Cardiovascular Sound

### KOROTKOFF SOUNDS

The Korotkoff sounds (1241-1286) are heard over arteries of the extremities immediately below a compressing cuff which is inflated to a pressure above systolic pressure and then slowly deflated. The Korotkoff sounds are used in determining aortic arterial blood pressure. The level at which they first appear is taken as the systolic peak pressure and the point of disappearance or marked change in tone as the diastolic level. The Korotkoff sound at the upper and lower level are what we usually think of as sound that is they are transients. At intermediate level of cuff pressure the Korotkoff sound are more murmurs. Korotkoff recognized three types in the sound: first five stages or tonal changes were described. The first phase is signalled by a tapping sound, the second by a softening of same, the third by a murmur, the fourth by a muffling of the sounds and finally the fifth by a total disappearance of sounds. It is the official recommendation of the American Heart Association (136) that the point of disappearance of sound be used as the most reliable index of diastolic pressure in most cases. It was stated that dulling of the sound (beginning of the fourth phase) is on the average 8 mm Hg above the true diastolic pressure. Systolic pressure is indicated by the first appearance of sound at 3-4 mm Hg too low on the average (136). The debate as to the most valid auscultatory index of diastolic pressure has raged from the earliest times (1019). In 1907 Pittenger (437) recommended disappearance in 1909 Fickler (439) recommended muffling. Since the report of Bordley *et al* in 1931 (136) recom-

mending disappearance Roberts, Smiley and Manning (1283) have made a strong case for the use of muffling. They based their recommendation on a sizeable series of intra-arterial pressure recording.

Recording of Korotkoff sounds have been made by several workers (601). Several other workers (319-615-1569) have used the Korotkoff sounds as the basis for an automatic blood pressure recorder (427A).

In some cases of systemic arterial hypertension and/or generalized arteriosclerosis and sometimes in aortic stenosis there is so-called an eulutory gap (1140). The sound may be heard between 240 and 200 and again between 150 and 120 with a silent period between 150 and 200 mm Hg. Occasionally a double gap, two silent periods may be found (1293). Rodbard and Crick (1294) found in auscultatory gap in cases of aortic stenosis and concluded that it corresponds to a plateau on the up stroke of the arterial pulse pressure wave. The gap could be widened by the application of a tourniquet to the forearm and eliminated by increasing blood flow with active hyperemia. In some cases of aortic stenosis the occurrence of this phenomenon was related to the presence of a deep inflection on the up stroke of the arterial pulse pressure curve.

In aortic regurgitation and in states of marked peripheral dilatation as in anemia, thyrotoxicosis or fever the Korotkoff sounds may be audible to zero pressure are audible without any compression of the artery. In none of these states is the diastolic pressure truly zero although it may be only 30 or 40 mm Hg. What one is hearing is

Paulin and Mannheim (1190) recorded a systolic murmur of at least faint intensity in 100 per cent of 108 children studied. In about half the cases intensity was sufficiently great that it was possible to classify the murmurs into two groups: (a) A "sinus-shaped" murmur separated from  $S_1$  by a short gap (the Still murmur) was present in 43 per cent of the 108 children. (b) A protosystolic decrescendo murmur was present in 93 per cent. Both types were recorded at the left sternal border but those of type (a) tended to be lower whereas those of type (b) were in the second inter-space.

Tromont and Gonin (492) suggested that an abnormality in the development of the infundibulum, such as relative narrowing, might be responsible for a functional murmur in many instances. They colorfully referred to the infundibulum as the "veritable crossroads" of cardiovascular pathology. In a similar vein is the familiar reference to the pulmonary artery as the "area of auscultatory romance," a designation which may have originated with Walsh (p. 23).

All seem agreed that the incidence of murmurs in newborns and in the first year of life (314) is very low but that the incidence increases steadily until in children and adolescents at least a slight murmur is demonstrable in the majority if not all. In a group of over 5000 newborns Richards (1270-1271) found an incidence of systolic murmur of only 1.7 per cent. The results of Lyons and his colleagues (999) were similar. The low incidence of systolic murmurs in the newborn increases the significance of any fetal murmur which may be heard or recorded before birth (see p. 205).

Murmurs in the newborn are in a few instances produced by congenital malformations: pulmonary stenosis and aortic stenosis are the particular deformities most likely to produce murmur from the beginning. Nadas (1137) states that of the systolic murmurs present at birth one in seven turn out to be on the basis of congenital heart

disease. Even a grade III or IV murmur present at the left sternal border at birth may disappear completely in one or two months. Richards (1271) found that a murmur heard at birth carries a 1/12 probability of persisting as an indication of congenital malformation. When a murmur is first heard at one year of age the chance of its representing a congenital malformation is only 1/50. Taylor (1463) found an incidence of 4.8 per cent for systolic murmurs among the newborn. In the majority the murmur had disappeared by one year.

Boone and Levine (133) followed up a group of patients, some of whom had murmurs labelled "organic," others with murmurs called "functional," depending on the basis of grade of intensity. It was found that cardiomegaly was ten times more frequent in the former type of case partially justifying intensity as a criterion of significance. Kuttner and Markowitz (830) on following up a group of cases after eight years found that 48 per cent of those originally thought to have "organic" murmurs had evidences of heart disease, whereas the figure was 13 per cent for the group with "functional" murmurs. In Australia Stuckey, Dowd and Walsh (1462) concluded that of every ten school children with a murmur one has rheumatic heart disease, two congenital heart disease and seven functional murmurs.

The reports in the literature of a murmur interpreted as that of ventricular septal defect disappearing later probably were cases of functional murmur. For example F. Purkes Wether (1516) in an article entitled "Can the clinical manifestations of congenital heart disease disappear with the general growth and development of the patient?" described a child in whom a very loud systolic murmur with thrill was found at 14 months and at 2 years but there was absolutely no murmur at 5 years or at 10 years and the boy was picked for the Royal Navy.

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mending disappearance Robert Smiley and Manning (1285) have made a strong case for the use of muffling. They based their recommendation on a sizeable series of intra-arterial pressure recording.

Recordings of Korotkoff sound have been made by several workers (601). Several other workers (319-613, 1369) have used the Korotkoff sounds as the basis for an automatic blood pressure recorder (1271).

In some cases of systemic arterial hypertension and/or generalized arteriosclerosis and sometimes in aortic stenosis there is a so-called "cullator" gap (110). The sound may be heard at us is between 210 and 200 and again between 150 and 120 with a silent period between 150 and 200 mm Hg. Occasionally a double gap two silent period may be found (1291). Redbird and Calkins (1291) found an auscultatory gap in case of aortic stenosis and concluded that it corresponds to a plateau on the upstroke of the arterial pulse pressure wave. The gap could be widened by the application of a tourniquet to the forearm and eliminated by increasing blood flow with reactive hyperemia. In some cases of aortic stenosis there were double Korotkoff sounds, the occurrence of this phenomenon was related to the presence of a deep minimum on the upstroke of the arterial pulse pressure curve.

In aortic regurgitation and in states of marked peripheral dilatation as in aneurysm, thyrotoxicosis or fever the Korotkoff sounds may be audible to zero, i.e. are audible without any compression of the artery. In none of the conditions is the diastolic pressure truly zero although it may be only 30 or 40 mm Hg. What one is hearing is



the pistol shot sound (see below). Usually there is to be heard a change in quality of the sounds when one goes from true Korotkoff to pistol shot sounds. It is customary to record the pressure in such instances as 200/40-0 mm Hg for example, 40 mm being the level of change in tone. Other peculiarities of the Korotkoff sounds: (1) Graud and Bert (554) described an apparent lowering of the diastolic pressure when the forearm was hyperextended. (2) Lim and Geismar (921) found that in cases of aortic stenosis even when associated with aortic regurgitation the Korotkoff sounds disappear 30 or 40 mm Hg above the point at which oscillometric fluctuations are minimal. This phenomenon is demonstrated by taking the blood pressure with an oscillometer rather than the usual device. Its discoverers recommended it for the demonstration of organic aortic stenosis.

Rodbard and his colleagues (1298) have recently shown by means of recordings that the lag period between events in the heart and the

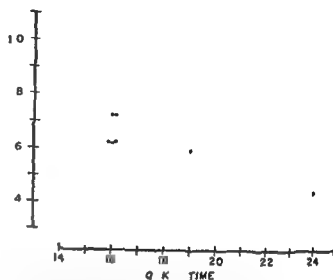


FIG. 239 The relation between the interval from onset of the QRS to the Korotkoff sound (abscissa in seconds) and the duration of the previous cardiac cycle (ordinate in seconds) in patients with atrial fibrillation (Courtesy of Rodbard and Margolis (1296) and *Circulation*.)

onset of the Korotkoff sound varies with the pressure in the cuff. As the pressure is lowered the QK interval, as they called the lag between the QRS and the Korotkoff sound, is shortened. Rodbard had shown earlier (1292) that the duration and intensity of the Korotkoff sounds afford an appraisal of the volume of blood flow to the extremity and that (1296) the time of onset of the Korotkoff sounds in the cardiac cycle is related to the diastolic filling time.

Ordinarily, in atrial fibrillation it is difficult even to estimate mean arterial pressure by the auscultatory method because of the variability in intensity of the Korotkoff sounds (Fig. 238). Rodbard and Margolis (1296) indicate that in atrial fibrillation the determination of the incidence of the sounds (percentage of EKG cycles with sounds) at various cuff pressure levels permits a satisfactory measure of the blood pressure level despite irregularities of the pulse. The QK interval in atrial fibrillation becomes shorter (Fig. 239) and the Korotkoff sounds louder with longer diastolic periods, probably because the velocity of the pressure pulse wave in the arterial tree is related to stroke output.

The Korotkoff sounds are weakest when the blood pressure is determined with the arm dependent (93). Also slow inflation of the cuff and redetermination of blood pressure without com-

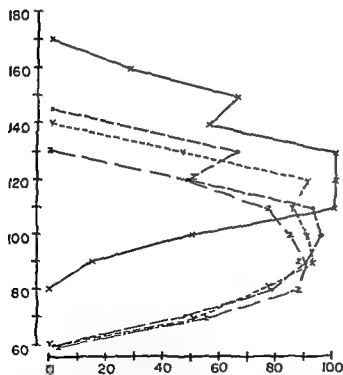
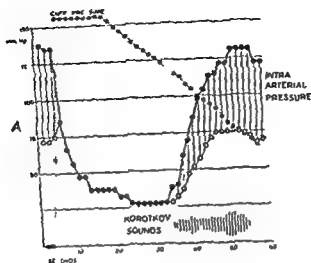


FIG. 238 In four subjects with atrial fibrillation the percentage of heart cycles (as indicated by the electrocardiogram) which had representation in the form of a Korotkoff sound at the brachial artery are plotted on the abscissa for different cuff pressures on the ordinate (Courtesy of Rodbard and Margolis (1296) and *Circulation*.)

RAPID INFLATION OF CUFF



SLOW INFLATION OF CUFF

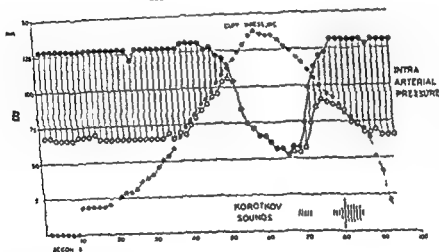


FIG. 10. Difference in Korotkoff sound with rapid and slow inflation of cuff.

With low inflation diastolic pressure is inflated in a manner that no sound is audible in an intermediate zone. In an aneurysm a gap is created. (From Rabin and Boriklev (1279).)

plete deflation of the cuff in the interval result in weak Korotkoff sound. An aneurysm gap (Fig. 240) is most likely to occur under the same circumstances (1279). The common denominator is thought to be venous engorgement. These are experiences further indicating that even though the level of blood pressure is not influenced the intensity of the Korotkoff sound is considerably dependent on the volume of blood flow to the area. In an open tube the volume of flow is dependent on the difference in pressure at the two

end of the tube. The same principle obtains in the arm. Elevation of venous pressure reduces blood flow to the arm by reducing the pressure differential between artery and vein. The report by Rodbard and Margolis (1291) of an aneurysm gap in arterio-sclerotic heart disease is consistent with the other observations and with the conclusion that the volume of blood flow is an important factor in the intensity of the Korotkoff sound. Reversible hyperemia eliminates the aneurysm gap. The gap can be induced in persons

not showing it spontaneously, by reducing total blood flow to the arm, for example, by occluding flow completely with a tourniquet of the forearm and determining the blood pressure in the conventional manner with the cuff on the upper arm. Amplification and recording of the Korotkoff sounds indicate that they are reduced greatly in intensity but do not disappear during the gap.

Galbruth (508) reported double Korotkoff sounds in one arm in a case of dissecting aneurysm of the aorta, presumably with double barrel configuration.

### THE PISTOL SHOT OR WATER HAMMER SOUNDS

The pistol shot or water hammer sounds are heard over peripheral arteries in aortic regurgitation and occasionally in other conditions in which peripheral vasodilatation is striking and/or stroke volume is large such as fever, anemia, pregnancy, and thyrotoxicosis. "Pistol shots" can be produced in normal individuals by administration of Apresoline or Priscoline or by inhalation of 10 per cent oxygen (839). The combination of increased cardiac output and peripheral vasodilatation is responsible. Generalized arterio-sclerosis, especially aortic sclerosis, favors the appearance of the phenomenon. In all of these conditions including generalized arterio-sclerosis there is a change in the shape of the arterial pressure curve which becomes steeper in both its aortic and carotid limbs with higher peak pressure and a generally narrow contour. The pistol shot sound is produced by the impact of this sharp pulse wave on the peripheral arteries. Qvistad and Steinert (1237) found a blood pressure of 200/110/50-60 mm Hg in a young woman with marked aortic calcification. "Pistol shots" tend to occur in such patients, despite the absence of aortic regurgitation.

As to mode of genesis the 'pistol shot' sound is probably fundamentally closely akin to the Korotkoff sounds discussed in the last section. The acoustical similarity is responsible for the fact that in aortic regurgitation and certain other conditions in which pistol shot sounds occur the diastolic blood pressure is usually given as "zero." The explanations which have been

offered for these sounds are several (557) (1) sudden expansion of the vessel wall with resultant vibration and sound production; (2) a "water hammer" effect due to sudden change in flow or pressure (433, 434, 814) (3) fluttering of the vessel wall due to Bernoulli effect (1292), (4) rapid change from one velocity profile to another (839).

### THE SIGNS OF DUROZIER AND TRAUBE

In aortic regurgitation and at times in the conditions which simulate aortic regurgitation as far as peripheral signs are concerned (fever, anemia, thyrotoxicosis, pregnancy, adrenaline administration), if the bell of the stethoscope is applied to a superficially located peripheral artery, such as the brachial in such a manner that moderate compression is applied, one may hear a double murmur. The first of these occurs in normal subjects although it may be exaggerated in persons with the above mentioned disorders, and is the usual systolic murmur heard with partial occlusion of a peripheral artery e.g., the Korotkoff sound. The second murmur is not usually present in the normal. The sign of Traube is a double sound (not a double murmur) heard over peripheral arteries in the same pathologic states as the Durozier sign but without pressure of the stethoscope. It is much less frequently present than the Durozier sign but the mechanism is probably closely akin.

It is the second component of the signs of Durozier and Traube which presents the main problem in genesis. It is possible that it is produced by backflow past the site of narrowing in the artery. Backflow of some degree is demonstrated in the femoral artery for example, by McDonald and his collaborators (668) under ordinary circumstances. Probably it is usually of insufficient degree to result in a murmur under the conditions accompanied by the Durozier sign it is presumably exaggerated.

On the other hand, Fausch (978) concluded that both sounds in the Durozier and Traube signs are produced by forward flow. His evidence was derived mainly from two facts: (1) distal

<sup>1</sup> Korotkoff him self subscribed to this view.

<sup>2</sup> See reference 975.

compression of the artery exaggerated the phenomenon (2) both the first and second elements were transmitted down the vessel with approximately the velocity of the pulse wave Blumgart and Lenné (123) thought backward flow was operative in aortic regurgitation but that in aneurysm fever and thyrotoxicosis and the situation when the arm is placed in warm water—states of peripheral vasodilatation—the sounds are produced with forward flow Blumgart and Lenné (121) based these conclusions on the observations (1) that in aortic regurgitation the second element (diastolic murmur) was accentuated by pressure on the distal edge of the sphygmograph bell placing the arm in cold water or applying a cuff inflated to a sub-diastolic level of pressure on the arm distal to the point of auscultation and (2) that in the non-regurgitation group of conditions the diastolic murmur is increased by pressure on the proximal edge of the sphygmograph and abolished by placing the arm in cold water or applying a cuff distal to the point of auscultation.

Hale McDonald and Womersley (623) observed two velocity peaks in forward femoral arterial flow assuming that these peaks are exaggerated in aortic regurgitation this observation may be evidence in favor of the mechanism proposed by Turova. On the other hand the observations of the same group on backflow in the femoral artery (663) might lend support to the mechanism advanced by Blumgart and Lenné for the Durosoz sign of aortic regurgitation.

#### FETAL HEART SOUND

The fetal heart sounds (197) consist of first and second sounds (Fig 241) as in the adult although one or the other may be quite faint. Third and fourth sounds have not been identified to my knowledge. Fetal heart sounds are best heard just above the symphysis pubis of the mother. Later the area of the abdomen will where the fetal heart sound are most clearly audible is determined largely by the position and presentation of the fetus. The rate is of course rapid (120-140 per minute). Fetal heart

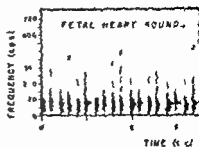


Fig. 241. Fetal heart sound.

sounds are usually audible at the fifth month early as early as the twelfth or fourteenth week.

Caustal diagnosis of congenital heart disease on the basis of a fetal heart murmur has been made by Simpson, McCulla and Kerr (1444), Pidgeon (1174), Dippel (333), Roberts (1242), Barry and Helman (92) and others (18). Ventricular septal defect is usually the lesion present (92, 1462). Congenital heart block has been diagnosed in a few instances on the basis of the rate of the heart sounds (77, 159). In one instance (1103) it was possible to establish that both fetuses were living by demonstrating two sets of fetal heart sound at different rates.

The number of workers who have applied their eases to the recording of the fetal heart sound is large (140, 61, 921, 921, 1040, 1216). By comparison the productivity has been slight. Corner and Strim (294) have used the fetal heart sounds as the triggering mechanism of a rate meter to use in studies of fetal distress. A proper time constant (0.20 sec.) inures that the meter is actuated only once per cardiac cycle.

Croom (603) demonstrated a systolic murmur in a recording of the fetal heart sounds and expressed the opinion that this would be entirely expected in all cases because of the patent foramen ovale and ductus arteriosus in the fetus. It is doubtful however that these would generate sound until septal defects per se do not in the adult the ductus arteriosus is large in these cases and has essentially a normal relation to the outflow of the right ventricle. The physiologic polycythemia of the fetus tends to counteract murmur production. In the fetal lamb twisted with placental connections intact a faint systolic

not showing it spontaneously, by reducing total blood flow to the arm, for example, by occluding flow completely with a tourniquet of the fore arm and determining the blood pressure in the conventional manner with the cuff on the upper arm. Amplification and recording of the Korotkoff sounds indicate that they are reduced greatly in intensity but do not disappear during the grip.

Galbruth (508) reported double Korotkoff sounds in one arm in a case of dissecting aneurysm of the aorta, presumably with double barrel configuration.

### THE PISTOL SHOT OR WATER HAMMER SOUNDS

The pistol shot or water hammer sounds are heard over peripheral arteries in aortic regurgitation and occasionally in other conditions in which peripheral vasodilatation is striking and/or stroke volume is large, such as fever, anemia, pregnancy, and thyrotoxicosis. 'Pistol shots' can be produced in normal individuals by administration of Apresoline or Priscoline or by inhalation of 10 per cent oxygen (839). The combination of increased cardiac output and peripheral vasodilatation is responsible. Generalized arteriosclerosis, especially aortic sclerosis, favors the appearance of the phenomenon. In all of these conditions, including generalized arteriosclerosis, there is a change in the shape of the arterial pressure curve which becomes steeper in both its anacrotic and catacrotic limbs with higher peak pressure and a generally narrow contour. The pistol shot sound is produced by the impact of this sharp pulse wave on the peripheral arteries. Quigstad and Steimert (1237) found a blood pressure of 200/210/50-60 mm Hg in a young woman with marked aortic calcification. 'Pistol shots' tend to occur in such patients, despite the absence of aortic regurgitation.

As to mode of genesis, the 'pistol shot' sound is probably fundamentally closely akin to the Korotkoff sounds discussed in the last section. The acoustical similarity is responsible for the fact that in aortic regurgitation and certain other conditions in which pistol shot sounds occur the diastolic blood pressure is usually given as zero. The explanations which have been

offered for these sounds are several (507): (1) sudden expansion of the vessel wall with resultant vibration and sound production,<sup>1</sup> (2) a 'water hammer' effect due to sudden change in flow or pressure (433, 434, 814), (3) fluttering of the vessel wall due to Bernoulli effect (1292), (4) rapid change from one velocity profile to another (839).

### THE SIGNS OF DUROZIEZ AND TRAUBE

In aortic regurgitation and at times in the conditions which simulate aortic regurgitation as far as peripheral signs are concerned (fever, anemia, thyrotoxicosis, pregnancy, adrenaline administration), if the bell of the stethoscope is applied to a superficially located peripheral artery, such as the brachial in such a manner that moderate compression is applied, one may hear a double murmur. The first of these occurs in normal subjects although it may be exaggerated in persons with the above mentioned disorders and is the usual systolic murmur heard with partial occlusion of a peripheral artery, e.g., the Korotkoff sound. The second murmur is not usually present in the normal. The sign of Traube is a double sound (not a double murmur) heard over peripheral arteries in the same pathologic states as the Duroziez sign but without pressure of the stethoscope. It is much less frequently present than the Duroziez sign but the mechanism is probably closely akin.

It is the second component of the sign of Duroziez and Traube which presents the main problem in genesis. It is possible that it is produced by backflow past the site of narrowing in the artery. Backflow of some degree is demonstrated in the femoral artery for example by McDonald and his collaborators (668) under ordinary circumstances. Probably it is a result of insufficient degree to result in a murmur under the conditions accompanied by the Duroziez sign it is presumably exaggerated.

On the other hand, Lunsford (978) concluded that both sounds in the Duroziez and Traube signs are produced by forward flow. His evidence was derived mainly from two facts: (1) distal

<sup>1</sup> Korotkoff has also subscribed to this view.

<sup>2</sup> See reference 978.

## SECTION IV

### *Cardiovascular Sound in Disease*

murmur produced at the patent ductus and transmitted down the aorta may be heard (137) The low incidence of murmurs in the newborn human (1270) increases the significance of any fetal murmur which is detected

The funic, umbilical or fetal souffle, a murmur generated in the umbilical cord and having the rate of the fetal heart, occurs fairly frequently

At times it is only systolic, in other cases continuous It is particularly likely to occur if there is twisting of the umbilical cord, for instance, around the neck of the fetus The rate differentiates it from the uterine, or maternal souffle Its changeable character aids in the difficult differentiation from a fetal cardiac murmur (355) Obviously the significance is quite different

## CHAPTER 15

# Valvular Heart Disease

The classification of heart disease followed in this discussion is in essence that suggested by Hamman (629) and more recently by Harvey (646) for use in considering the causes of heart failure: valvular heart disease, myocardial disease, pericardial disease, systemic hypertension, pulmonary hypertension, congenital heart disease and miscellaneous disorders not properly placed in any one of the previous categories.

In the case of disease of each valve, stenosis and regurgitation may be the functional consequence of impairment of normal valvular functions, i.e., to open allowing forward flow with a minimum of obstruction and to close without back leak.

Rheumatic fever remains the leading villain in the causation of valvular disease. The order of frequency of involvement of the four heart

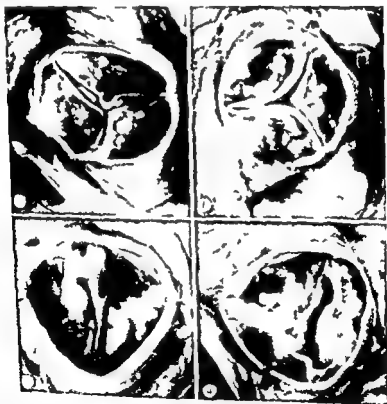


FIG. 247 Anatomic grades of calcific aortic stenosis.

Photograph of the valve from the aortic side. Grade I (a) moderate rigidity and calcification of the aortic cusps with minimal or absent commissural fusion. Grade II (b) moderate rigidity and calcification with fusion at one commissure. Grade III (c) marked rigidity but still with some flexibility, marked calcification, fusion of two commissures. Grade IV (d) pronounced calcification and rigidity without flexibility, fusion of at least two commissures, minimal residual oration. (From Anderson, Kelcey and Edwards (21).)





valves is the same as the order of pressures which the valves must sustain in the closed state (877-1479). From the lower incidence to the higher the order of valve involvement is pulmonary (11 per cent), tricuspid (19.6 per cent), aortic (33 per cent), and mitral (56 per cent), the corresponding pressures to which each of the valves is exposed at closure are approximately 3-24-72 and 116 mm Hg.

#### AORTIC STENOSIS (15)

**ETIOLOGIC AND ANATOMIC CONSIDERATIONS** (724) The long controversy between the two schools—one of which advocated atherosclerosis as the cause of calcific aortic stenosis and the other which advocated rheumatic fever (768) has been satisfactorily resolved by the concept (which has much to support it) that rheumatic fever is one of the processes which, by damaging the heart valve can prepare the soil for atherosclerotic change and calcification (126). Congenital bicuspid state of the valve is another (1404) and congenital aortic stenosis which is now known to undergo calcification is a third. This multifactorial or at least bifactorial basis of calcific aortic stenosis accounts well for the fact that isolated aortic stenosis occurs predominantly in men in women mainly after the menopause in diabetic and in idiopathic hypercholesterolemia.

Calcific aortic valve disease occurs usually often in association with Piguet's disease of bone (725B).

Various classifications of the grades of aortic stenosis can be set up (21). The mildest grade is that variety in which the individual cusps are rendered rigid by fibrosis and calcification, but there is little or no fusion at the commissures. The severest grade is that in which complete fusion has occurred with only a small opening in the center (Fig. 242).

Both valvular and subvalvular (subaortic) varieties of congenital aortic stenosis occur as well as combined forms. Although the subaortic variety is of course easily identified at autopsy and is clearly congenital the problem of differentiating congenital from a partly acquired variety can be difficult not only clinically but also pathologically especially in late stages when secondary calcifying atherosclerotic changes have taken place. Description of a murmur from an early age—at least as early as four years by the criterion of some—is taken as indication of congenital bicuspid. Poststenotic dilatation of the aorta occurs in all types of aortic stenosis and occasionally attains mammoth proportions (1081). Logue, Robinson and I (1081) described

FIG. 243. Aortic stenosis with dissecting aneurysm of the ascending aorta.

Case 1. A white male student of the age of 27 had been known to have a heart murmur since the age of 3. There was no history of rheumatic fever and he had never had symptoms referable to the cardiovascular system. In 1945 an x-ray of his chest showed dilatation of the ascending aorta with a heart of normal size (Fig. 243 top left). On the day of admission he suddenly developed severe constant dull aching sub-sternal pain extending from the epigastrium to the neck. The pain increased in severity and he was admitted to the student infirmary.

Physical examination showed no evidence of the Marfan syndrome. The blood pressure was 106/84 mm Hg in both arms; it was not recorded in the legs. The heart was normal in size. The cardiac rhythm was regular. There was a harsh grade III aortic murmur accompanied by a systolic thrill at the first and second right intercostal space. The aortic second sound was replaced by a faint early high pitched blowing diastolic murmur. The aortic murmur was transmitted over the precordium toward the apex. The first sound at the apex was normal. The aortic murmur was not transmitted to the axilla. No diastolic murmurs could be heard at the apex. The lungs were clear and resonant. Lungs and by ten were not tender or palpable. The remainder of the examination was normal. An electrocardiogram taken the day after admission showed left ventricular hypertrophy. On the following day the electrocardiogram showed changes consistent with acute pericarditis. During his hospital stay the patient continued to complain of chest pain requiring opiates for relief. During the following 3 days the pain became less severe. 4 days after admission however there was an increase in severity of chest pain associated with sudden gasping respirations that continued for 10 minutes when he died.

Autopsy showed aortic stenosis (B top right and bottom) with some calcification of the valve and with left ventricular hypertrophy. There was no evidence of rheumatic involvement of the mitral valve and the aortic stenosis was thought to be of congenital origin. There was a dissecting aneurysm which began within the first few centimeters of the aortic valve in the area of dilated aorta. There was retrograde extension of the dissection with rupture into the pericardial sac. Death occurred as a result of tamponade. Macroscopic examination showed cystic

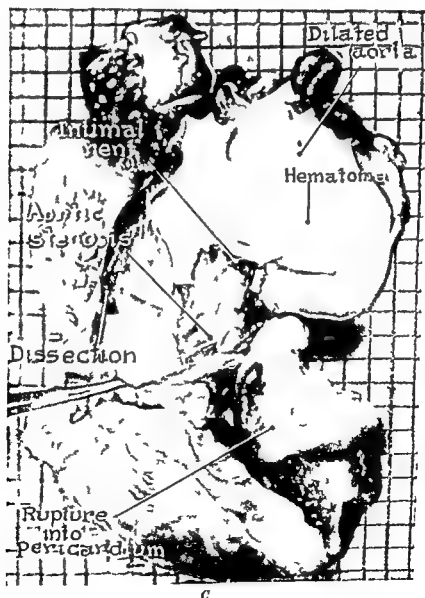
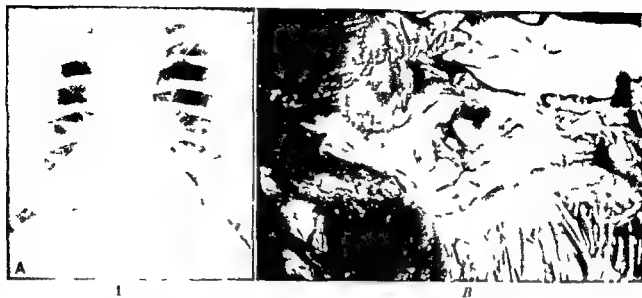


FIG 213 1 B and C

four cases of aortic stenosis and regurgitation in which cystic medial necrosis and dissecting aneurysm occurred in the ascending aorta (Fig. 243 and 244)

**PHYSIOLOGIC CONSIDERATIONS** Were it not that pressure proximal to a stenotic aortic valve is increased mean pressure and flow in the systemic circulation would not be maintained at level compatible with life. Probably a systolic pressure differential of more than 150 mm Hg between ventricle and aorta rarely exists even in instance of most severe aortic stenosis. Corlin and colleagues (378) found 150 mm Hg the highest gradient in three patients whom they studied. The limiting factor seems to be ventricular systolic pressure which seldom exceeds 200 mm Hg. This ceiling, in turn imposed by the coronary flow necessary for the increased ventricular work. The interrelationship of pressure, flow and valve area shown by these authors (378). A fixed stroke volume limit in a patient with pure AS in the absence of gross failure and often even in the absence of symptoms may indicate a tiny aortic orifice (378).

Valve orifice area (378) can be calculated—provided there is no appreciable aortic regurgitation—by a modification of the formula used for a similar purpose in the case of mitral stenosis. Data of left heart catheterization are necessary.

Corlin's formula is applicable only to mild orifice stenosis. Its validity cannot be tested in the normal valve orifice. The reason is that the constant in the formula derived empirically is in fact variable. The value of this constant is somewhat dependent on the form of the orifice (1303).

It was discovered as anticipated after application of clamps and ligation of the aorta that in fact occurred with a characteristic sheath like double channel in the ascending aorta. The false channel was traversed by typical filiform coils. The surgical procedure was performed according to plan without evidence of cardiac embarrassment. Soon after the clamp was removed from the aorta pulsations of the heart became weak and ventricular fibrillation began. Cardiac massage and other efforts at resuscitation were of no avail.

At autopsy the heart weighed 900 gm, the excess weight being mainly the result of left ventricular hypertrophy. There was calcific aortic stenosis with fusion of one commissure and the valve appeared regurgitant (Fig. 245). In aortic dissection extended 15 cm above the aortic valve through the end of the aortic arch and into the innominate and subclavian arteries. The thoracic aorta was of normal size. Microscopic study showed cystic medial necrosis in the ascending aorta and to much less extent in the subclavian, mesenteric and pulmonary arteries and in the abdominal aorta.

$$A_1 = \frac{CO/SFP}{41.3 \times \sqrt{L_{1M} \times R_{1M}}}$$

where  $A_1$  = aortic valve area

$CO$  = cardiac output

$SFP$  = systolic pressure in sec/mm

$L_{1M}$  = systolic mean pressure in the left ventricle

$R_{1M}$  = systolic mean pressure in the brachial artery

Symptomatic pure aortic stenosis is usually found to have a valve orifice area of about 0.5 cm.

In all valvular stenoses a more meaningful index with reference to murmur production might be the valve resistance calculation by Pourcil-Lewis used by Dow and colleagues (370). This calculation takes both pressure gradient and flow into account.

$$VR = \frac{(L_{1M} - L_{2M}) \times 1332}{CO}$$

where  $VR$  = aortic valve resistance in dyne/sec/cm<sup>5</sup>

$L_{1M}$  = mean left ventricular pressure in mm Hg

$L_{2M}$  = mean aortic pressure in mm Hg

$CO$  = cardiac output in cc/sec (This value must include regurgitant volume if there is any, i.e., it must represent total cardiac output.)

The shape of the aortic pressure curve is altered in a characteristic manner in aortic stenosis. The peak is delayed with the amount of delay being proportional to the severity of the aortic stenosis. Studies in models (244) indicate



1



B

*The 24 Aortic stenosis with dissecting aneurysm of the ascending aorta*

T. G. M. (726673) a 50 year old attorney during his youth in a southern state had numerous episodes of acute tonsillitis but recalled no definite rheumatic fever.

At the age of 24 years he was first told that he had mitral stenosis. At the age of 35 years he was twice rejected for service in the Merchant Marine and in the Army because of murmurs. He was still asymptomatic. Intermittent dyspnea had its onset at the age of 43 years and soon thereafter attacks of pyrexia and nocturnal dyspnea developed. Aortic aneurysm was discovered when he was 48 years old. Chest pain was never a conspicuous feature.

Physical examination revealed a blood pressure of 190/91 mm. Hg in both arms. 235/105 in both legs. There was an active expansive systolic pulsation in a large area below the right clavicle and the same area was dull to percussion. In this same area furthermore there was a grade IV systolic murmur accompanied by a thrill and followed by a decrescendo diastolic murmur typical of aortic regurgitation. What seemed to be the same diastolic murmur was heard out toward the right axilla where it acquired a delicate high pitched quality down the left sternal border and in the apical area where its quality was more low pitched and rumbling. The heart was strikingly enlarged to the left. Atrial fibrillation was present.

In general nothing about the patient's habitus or family history suggested the Marfan syndrome. The ophthalmologist could find no evidence of ectopia lentis.

Roentgenograms showed a large aneurysm of the ascending aorta and considerable enlargement of the left ventricle.

An operation similar to that used in the first patient was planned and at thoracotomy an enormous aortic aneurysm

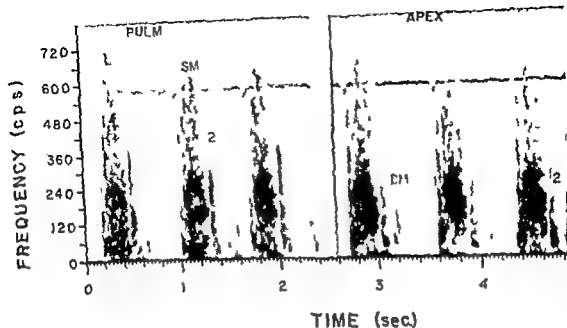


FIG 24 Full blown rheumatic aortic stenosis at the age of 7 years (VERGIL) was first seen at 3½ years with acute rheumatic fever manifested by pericarditis, changing murmurs, congestive failure, and closing of rheumatic fever continued thereafter. At the base (left) there are typical findings of aortic stenosis. At the apex (right) the aortic murmur is transmitted from the base and there is an early diastolic murmur which is probably of mitral origin since it begins after S by a short gap.

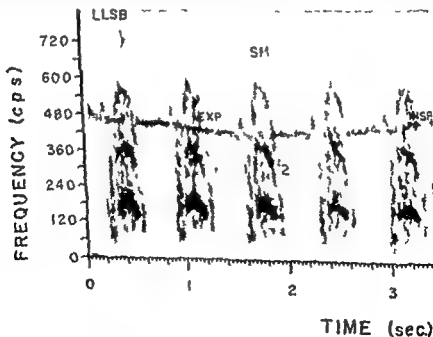


FIG 28 LLSB in P.M. (MISH) with aortic stenosis. Both noise and musical elements are present with the latter predominant in this area. There is paradoxical splitting of the second sound which was better shown in the recording from the pulmonic area. The pulmonary closure sound is at the end of the murmur which stops slightly before the aortic closure sound.

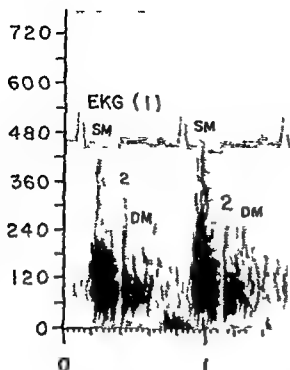


FIG. 245 Aortic area in aortic stenosis and regurgitation on rheumatic basis. Note Christmas tree configuration of systolic murmur gap between murmur and second sound (2) diastolic murmur decreases in both intensity and peak frequency.

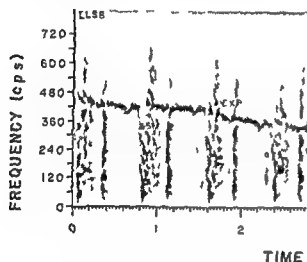


FIG. 246 Mild aortic stenosis.

C B R (231784) age 54 had palpitations at age 11 and was kept at bed rest for most of a year. She had been plagued by tonillar troubles most of her life. The heart was at the upper limit of normal for size. EKG showed left axis deviation and left ventricular hypertrophy. A somewhat scratchy systolic murmur is present in the aortic area and transmitted into the base of the neck on the right. The murmur stops well before S. There is a pre-systolic gallop consistent with aortic stenosis. The murmur has the appearance associated with scratchy sounds. This is probably mild aortic stenosis.

that the change in the arterial pulse pressure curve in aortic stenosis is not explicable on the basis of the stenotic orifice *per se*. Instead, the basis must be sought in a change in the pattern of ventricular contraction.

The end diastolic pressure in the left ventricle is elevated in cases of marked aortic stenosis, and left heart catheterization usually reveals giant a waves in the left atrial pressure pulse (62). Hypertrophy of the left ventricle undoubtedly results in change in the pressure volume characteristics of the ventricle and probably is responsible for the elevated end diastolic pressure in the absence of heart failure in the usual sense. The giant a waves, elevated end diastolic left ventricular pressure and the pre-systolic gallop of aortic stenosis are probably causally interrelated phenomena.

In the case of both ventricles functional outflow obstruction caused by encroachment of hypertrophied myocardium on the outflow tract is being recognized with increased frequency. By left heart catheterization Brock (1701) finds a gradient across the aortic valve area in cases of long standing systemic arterial hypertension. This phenomenon represents a basis for an aortic systolic murmur in cases of hypertension. Hancock *et al* (635) also write about 'pseudo-stenosis' of the aortic valve in patients with a murmur typical of aortic stenosis associated with a thrill in two of five cases. The basis for the murmur is not clear since left heart catheterization showed no 'measurable systolic gradient across the aortic valve'. One certainly must agree that there are cases with the murmur and thrill of aortic stenosis but no aortic obstruction of physiologic significance. However the production of these physical signs in the absence of any gradient whatever across the aortic valve is puzzling. Possibly in some cases fibrotic and rigid cusps without commissural adhesion vibrate vigorously in the outflow stream.

**CARDIOVASCULAR SOUND** Usually the systolic murmur characteristic of aortic stenosis is loudest in the aortic area and is often accompanied by a systolic thrill although a thrill on the one hand is not essential to the diagnosis and on the other hand, may occur with relative aortic stenosis (see below). Occasionally especially in children

the diagnosis is of aortic stenosis. Also the murmur is often well transmitted to the cardiac apex thereby creating possible confusion with mitral regurgitation (Fig. 247 and 249). The latter is such a frequent occurrence that special emphasis is indicated (36, 322). At times the murmur is audible only at the apex (89). The shape of the murmur in the PCG (see p. 194) and the fact that it is nonholosystolic—being separated from S by a brief silent interval—permits differentiation from the murmur of mitral regurgitation.

Occasionally, especially with rupture of the posterior chordae tendineae of the mitral valve, the murmur of mitral regurgitation may be loud in the aortic area and even over the carotid arteries resulting in simulation of aortic stenosis (see p. 600). There may even be a thrill in the aortic area because of the peculiar direction of the regurgitant jet from the mitral valve.

Occasionally there is an interval between the first heart sound and the onset of the systolic murmur (e.g. Fig. 249). Possibly this is more likely to be the case when left bundle branch block is present.

Usually the quality of the murmur of aortic stenosis can be described as harsh. Frequently it is musical (Fig. 248 and 249). When this is the case three patterns may be seen. There may be a poorly transmitted musical murmur in the aortic area. In older diabetic patients the appearance and evolution of a musical systolic murmur of this type may be observed on repeated examinations over the years. A second pattern which may be merely a later stage of the first is the presence of a very loud musical systolic murmur heard over the entire chest and transmitted into the neck and even the extremities. A third pattern is diagrammed in Figure 176. In these patients one hears in the aortic area and at the base of the neck on the right a conventional noise murmur. But at the left sternal border over the left midprecordium and at the apex the murmur is purely musical. This description is conveniently referred to as the Gallavardin phenomenon. The explanation offered for this situation is as follows: the noise murmur probably represents the jet of the musical murmur, the regular vibrations of the aortic valve diaphragm. Since the aortic valve underlies the left sternal border and since the tense contracting, hypertrophied left ventricle makes more intimate contact with the chest wall during systole—when the murmur is occurring—the radiation of the musical murmur is not surprising.

In 1897 Dickinson (33) wrote on the occurrence of musical mitral murmurs in connection with aortic stenosis. One of his cases may have been an instance of extracardiac musical murmur on the basis of massive cardiomegaly and/or pericardial effusion. Another of his cases may have represented unusual transmission of the aortic murmur to the apex. However, he suggested another mechanism for a musical murmur with aortic stenosis. In one case the aortic stenosis was so severe that peripheral pulses were not palpable and at autopsy water would flow through the orifice only in drops or small trickles. The mitral valve was normal. Dickinson suggested that the marked elevation of left ventricular pressure which was undoubtedly present during systole forced the normal mitral valve with production of a very small leak and a very high pitched murmur which he compared to the squeak of a mouse or guinea pig. He was again, however, of musical murmurs produced by other mechanisms. There may be musical murmurs of many kinds as there are many sorts of musical instruments. It will be difficult to prove the Dickinson mechanism for a musical aortic systolic murmur in aortic stenosis transmission of a musical form of the primary murmur seems most likely in such cases including Dickinson.

There are some cases of calcific disease of the aortic valve in which very intense and highly musical systolic murmur is heard everywhere (the second type mentioned above) with none of the discussion between the noise murmur and the musical murmur—in fact with little or no element. In these cases the evidence of aortic obstruction in terms of the pulse pressure and symptoms is usually minimal or it is most moderate. In such case it is likely that there is fibrosis and calcification of the valve cusps with little adhesion at the commissures (Grade I or II of Fig. 212). There is therefore no jet, but a very intense murmur is produced through vibration of the thickened cusps. Stokes (see p. 22) in 1833 was early to point out the paradox between murmur and clinical state in an old gentleman who complained that his entire body was one humming top although his general health continued excellent. In 1932 Schmidt (1360) described an asymptomatic 83-year-old farmer with calcific aortic stenosis and a systolic murmur audible 15 cm from the chest. The physical



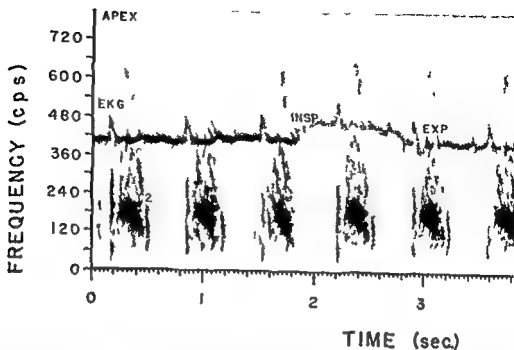


FIG. 219 Calcific aortic stenosis

Apex in N. G. (32028) 74 year old male with gout. He had been thought to have mitral regurgitation (probably with calcified valve because the murmur is musical). The character of the murmur especially the shape and frequency level of the harmonics and the gap before  $S_2$  (which is probably aortic closure sound in this recording at the apex) are most consistent with calcific disease of the aortic valve.

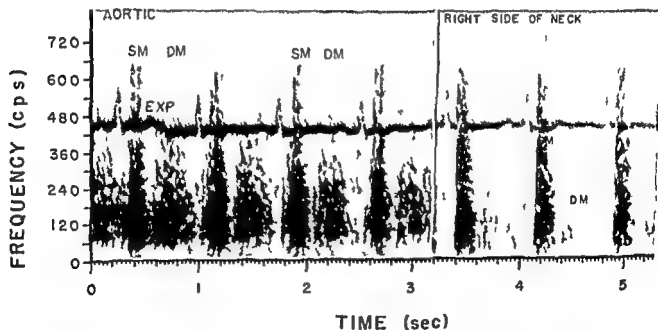


FIG. 220 Typical AS-AR with transmission to the neck

In L. H. B. (701509) 44 years old the findings in the aortic area (left) are completely characteristic. At the base of the neck on the right (right) the systolic murmur is late in onset consistent with the view that the systolic murmur travels at the velocity of the pulse wave. The diastolic murmur is also faintly demonstrated.

and especially with the subaortic variety of aortic stenosis the area of maximum audibility may be at the left costal margin. In such instances, ventricular septal defect may be a mistaken diagnosis.

Customarily, the murmur is well transmitted into the base of the neck (Fig. 250) and to the suprasternal notch. In fact, in the absence of this feature there is reason to be suspicious of

cause of the fibrotic and calcific process which is likely to have occurred in the valve the second sound may be at least normally intense in spite of a reduced range of motion. Although in general the aortic closure sound is reduced in very severe aortic stenosis this feature is not sufficiently constant to be helpful in much less severe cases to the diagnosis. Occasionally the second sound displays paradoxical splitting (p. 164) with the aortic component following the pulmonary and with the split greatest in expiration.

Pre systolic gallop occurs commonly with aortic stenosis as with other varieties of systolic overload of the ventricle. Aortic regurgitation may of course be associated with aortic stenosis in fact more often than not at least some light degree of aortic leak can be detected.

See page 358 for a discussion of certain special features of congenital aortic stenosis.

# AORTIC INSUFFICIENCY (AI)

(Syn. Aortic incompetence in efficiency; inadequacy, etc.)

ETIOLOGIC AND ANATOMIC CONSIDERATIONS (1972) The main cause of aortic valvular damage leading to predominant aortic regurgitation are rheumatism and syphilis. In addition dissecting aneurysm, Myer-Simpson's syndrome, the Marfan syndrome, idiopathic cystic medial necrosis of the aorta, hypertension, atherosclerosis, calcification of the aorta, congenital bicuspid aortic valve, other congenital malformations of the valve including ventricular septal defect with retroversion of the aortic cup (see p. 762), bacterial endocarditis, and trauma are factors which alone or in combination may lead to regurgitation at the aortic valve.

Severe predominant aortic regurgitation on a rheumatic basis occurs more commonly in men. Bland and Wheeler (114) found this situation to be twice as common in men. Review of cases of aortic regurgitation presenting for the Mufson operation turned up an unexpectedly large number of cases which appeared to be on a rheumatic basis. Previous history of a febrile illness, rheumatic fever, or arthritis in aortic regurgitation as an isolated or overwhelmingly predominant lesion most cardiologists would have expressed the opinion that this is rare.

Cydenman (216) described a case of marked fenestration of the aortic valve, with histologic evidence of cystic medial necrosis. Matthews and Driscoll (1011) had a rather similar experience. Their patient probably had slight rheumatic involvement of the mitral and aortic valves and had had signs of aortic regurgitation before the development of an abrupt change in the diastolic murmur to one with a high pitched whirring quality audible without a stethoscope. The patient died about 70 days after developing this murmur.

The particular affection of the aortic valve known as retroverted cup (Fig. 222) and is accompanied by a musical murmur is most often caused by syphilis. In 1939 Bell and colleagues (52) demonstrated that in syphilis loss of the fibrous skeleton on the ventricular surface of the aortic cup permits retroversion (Fig. 222). Furthermore it is usually the right anterior coronary cup which becomes retroverted.

In the literature there is at least one autopsy proved instance of retroversion of an aortic cup on the basis of cystic medial necrosis of the aorta (658). A second probable case is described (270) and I am following a patient who appears to represent a third (M. H. 701542). In the last patient a few old men a very loud aortic diastolic murmur of striking musical quality was audible to both the patient and his wife. No murmur of any variety was present on any of numerous previous examinations and the patient has remained essentially asymptomatic during more than three years of observation. Most of the musical murmur. Serologic tests for syphilis including treponemal immobilization test are negative and the aorta is not dilated. Matthews and Driscoll (1011) described spontaneous rupture with fenestration of the aortic valve apparently also on the basis of cystic medial necrosis.

Excessive straining and blunt trauma to the chest may be the immediate precipitating factor in retroversion of an aortic cup or in rupture in the case of fenestration or laceration of the attachment of a cup. A classic case is that described by Jones and Lindholm in 1879. Immediately after over exerting in connection with expulsion of an intruder to his home the patient noted a cooing in his chest. It was audible also

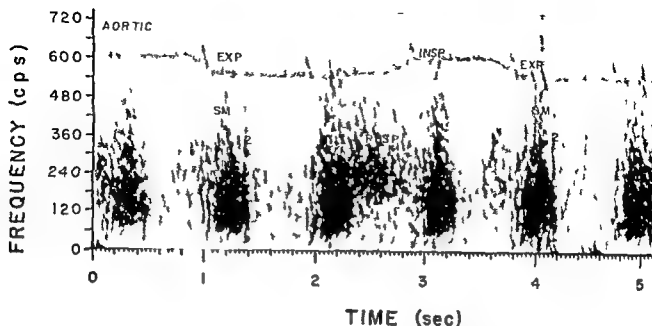


FIG 2a1 Effect of operation for aortic stenosis

R S (734185) a 50 year old white male had been having dyspnea, substernal tightness and syncope. On April 21 1956 under hypothermia the aortic stenosis was surgically treated under direct vision. The main obstruction was produced by fusion of the two coronary cusps. One year after operation (see fig. 2a1B) the peak of the systolic murmur was much earlier. A minimal murmur of aortic regurgitation was demonstrated. (Much background noise in A)

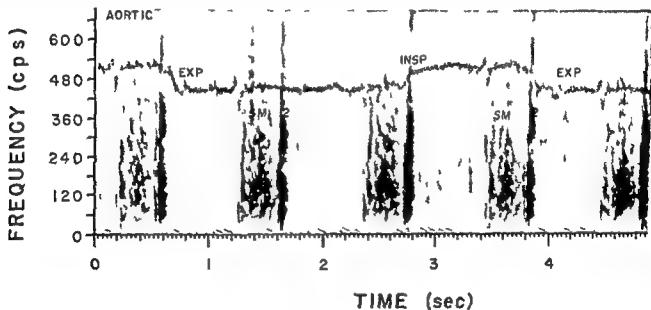


FIG 2a1B

can first heard the murmur when examining the fully clad patient with the ophthalmoscope.

The shape of the murmur of aortic stenosis is characteristic. In the oscillogram it is diamond shaped because of the location of its intensity peak in mid systole. In the spectrogram it is shaped like a Christmas tree because of its frequency peak in mid systole. Furthermore the

murmur of aortic stenosis stops before the aortic closure sound. There is usually a brief silent gap between the end of the murmur and the aortic component of the second sound. The shape of the murmur of aortic stenosis has adequate hemodynamic explanation (see Figure 98).

The second sound in aortic stenosis may be increased, normal or reduced in intensity. Be-

to his wife Autopsy revealed in addition to retroversion of the right anterior coronary cusp of the aortic valve typical intimal changes of syphilis in the ascending aorta - a syphilis was probably the underlying cause with trauma the immediate precipitating factor These mechanical factors operate in precipitating or exaggerating aortic regurgitation in the Marfan syndrome

A convincing case of aortic valve rupture due to trauma was reported by Leonard Harvey and Hufnagel (873) in a young man who was kicked in the chest by a trolley. In this patient rupture of the interatrial septum may also have occurred as suggested by the murmur and by the findings of two cardiac catheterizations. The possibility of rupture of a sinus of Valsalva into the right ventricle is an alternative possibility which would account for all findings. Usually when traumatic rupture of an aortic valve occurs there is pre-existing aortic rheumatic affection bacterial endocarditis or the Marfan syndrome.

In 1977 the typical grown appearance of the Chinese was not generally recognized. MacCallum (1982) wrote a follow-up to his 1957 review and a revelation to L. C. U. (1982) at the meeting of the German and logical sciences to the discussion by (Huan and others) of the theory by (Huan) and (Huan) of the spiritual nature of change and particularly to realize the perfect form of the (Huan) and (Huan) appearance of the world as it is. Since then everyone has recognized it as a simple fact to be one of those simple triumphs of the great that make one's mind to have been a

Prondit and McCormick (1215) described an instance of aortic valve rupture in a hypertensive man who was in an automobile accident. Levine, Koons and Clark (797) reported two convincing autopsied cases of traumatic rupture of the aortic valve. In 1928 C. P. Howard (711) was prompted to his case review of traumatic aortic regurgitation by the case of a 44-year-old chauffeur who developed a thrill in the chest following the strain of cranking a car. Two Wassermann reactions were negative.

Mazzetta (1941) described a 47-year-old man who was killed in the chest during a fight. Three months later a grade 3 diastolic murmur audible all over the chest was loudest at the apex. The patient died in heart failure nine months after the accident. At necropsy the writer showed a T-shaped tear just above the posterior cup of the aortic valve. A fistula-like passage extended from there to a point just proximal to the innominate orifice. I have observed a similar case in a 46-year-old man who developed aortic regurgitation after a load of brick fell on his chest (J. M. Hall 1952).

I have seen one patient (M. G. 21407 int. 22007) in whom a hysteric aneurysm at the base of the aorta ruptured behind the posterior aorta cup into the left ventricle producing sign of aortic regurgitation (2.8 Fig. 6).

\* Intertriginous (or stromectrin) probably can't such if ever be assigned as the primary cause of rupture of an aortic aneurysm, productive of a non renal dialysis murmur. One case reported a week in med (2'9) of non traumatic rupture of the aortic valve actually had syphilis. The pr

Fig. 25. Retroverted right anterior coronary aortic cu p. caused by syphilis. I felt it represents a "kissing" of the coronary cu p. due to the separation of the c. mm. w. p. tree marking of the aortic intima, relatively high position of the coronary cu p. The right anterior coronary cu p. is involved. B. Photograph of specimen of aortic atherosclerosis. W. H. J. H. 190000. A 31 year old virgin male was a patient in the hospital because of mild pulmonary edema. Syphilis and treatment for same were of mild six week before admission. At that time the patient was first seen in the I. M. Service. Discharge because of pulmonary edema. At that time a low diastolic pressure of 60 mm. Hg. of non-arterial quality was present. (Pressure was 140/40 mm. Hg. when it was again admitted to the hospital. At that time pressure was 170/70-80 mm. Hg. and the diastolic minimum was now the typical low diastolic or sea gull sound.)

rays revealed pronounced enlargement of the left ventricle with no evident aortic dilatation. Serologic tests for syphilis were positive and the final diagnosis of aortic aneurysm with fixation resection for syphilis was a positive. Numerous cultures were sterile and the patient remained afebrile.

The patient's clinical progressively more severe manifestations of congestive failure and died 11 months after appearance of the systolic murmur. X-rays reveal retroversion of the right anterior coronary cusp of the aortic valve and relatively little change in the other cusps. The width of the aortic valve corresponding to this cusp was 1.5 cm (1.5 cm) as were all other annuli to a lesser extent. The coronary arteries were slightly separated and the coronary ostia somewhat high but normally patent. Mild intimal changes typical of aortic atherosclerosis were present throughout the thoracic aorta.

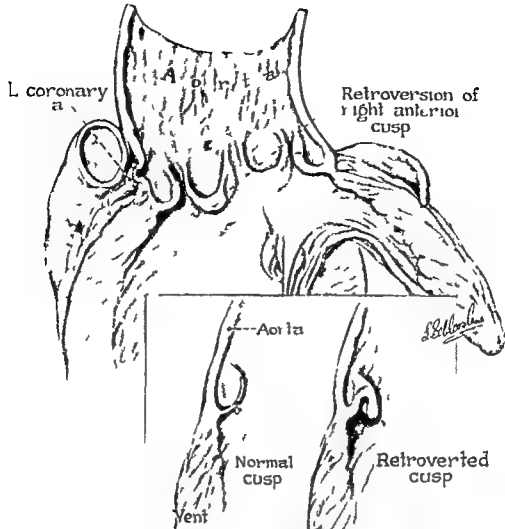


FIG 252 I

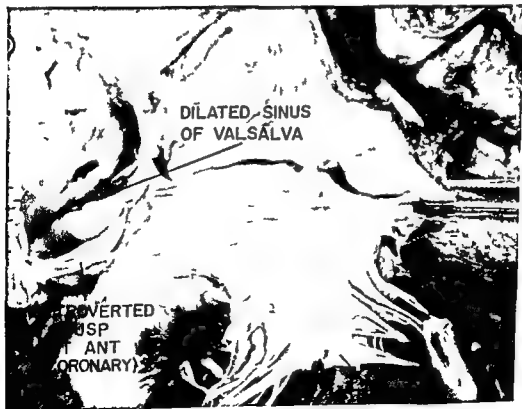


FIG 252 B



FIG. 53 Fenestration of semilunar cu (Aortic (above) and pulmonary (below) valves in 15-year-old man with congenital pulmonary Aortic to diastolic murmur was heard at the left sternil border (Courtesy of Fine and Hathaway (1961) and the American Journal of Medicine)

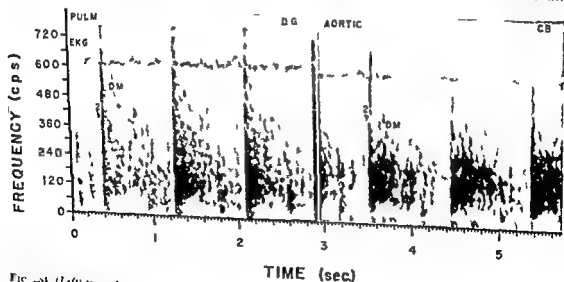


FIG. 54 (Left) typical aortic regurgitation (rheumatic) in pulmonary area. An aortic diastolic murmur was heard within three weeks of the onset of what was thought to be the first attack of ARF at age 10 in D.C. (40S-87). The patient was asymptomatic at age 21 and participated actively in sports as a basketball coach. (Right) typical aortic regurgitation (rheumatic) complicated by AS. Aortic area in C.B. (32-73)

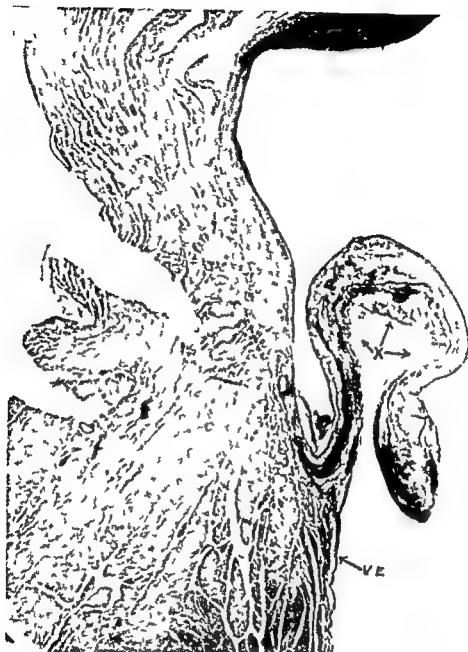


FIG. 25 ( Retroverted aortic cusp )

Destruction of the elastic fibers which sweep up onto the ventricular aspect of the valve and normally represent a retaining basket is demonstrated (From Bellet, Gouley, Nichol, and MacCallum (52) )

tient, a Negro laborer, was seen at the Johns Hopkins Hospital in 1901 by Drs. William Osler, Rufus Cole and Thomas McCrie. There was musical aortic diastolic murmur which Dr. McCrie compared to the bass string of a cello and which was audible six inches from the body. The autopsy was performed by Dr. William C. MacCallum who rendered the final diagnosis of rupture of an aortic cusp due to arteriosclerosis. (It was recorded. In connection with the musical murmur it is of interest to note the finding of

a cusp of the aortic valve which flapped back and forward and was probably the causative factor.) However by the statement of MacCallum himself (see footnote p. 271) syphilitic aortitis was not recognized in 1901. Furthermore recent review of the histologic sections reveals absolutely typical syphilitic aortitis. So-called rupture of the aortic valve may occur in the course of bacterial endocarditis.

Encysted semilunar valve tumors were found in 72% of 342 hearts by Friedman and Huthwaite



FIG 2b3 Penetration of semilunar cusp

Aortic (above) and pulmonary (below) valve, infarcted man with cor pulmonale. A grade 2 diastolic murmur was heard at the left sternal border (Courtesy of Friedman and Huthwaite (1964) in the American Journal of Medicine)

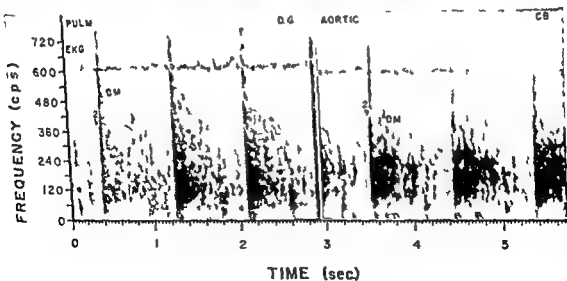


FIG 2b1 (Left) typical aortic regurgitation (rheumatic) pulmonary area. An aortic diastolic murmur was heard within three weeks of the onset of what was thought to be the first attack of ARF at age 10 in D.C. (40°N). The patient was asymptomatic at age 21 and participated actively in sports as a basketball coach. (Right) typical aortic regurgitation (rheumatic) complicated by SBE. Aortic area in C.B. (52°N)



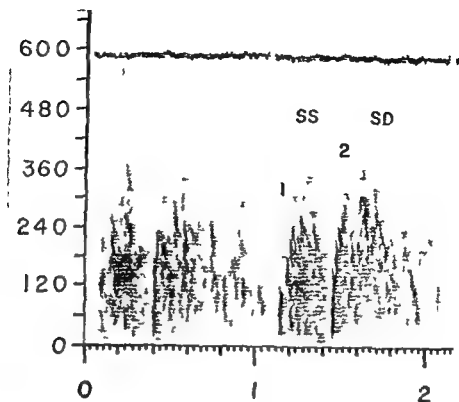


FIG. 2a5 IISB in case of typical aortic stenosis and regurgitation. There is a gap between the end of the Christy murmur (SS) and S. The diastolic murmur (SD) is briefly crescendo then decrescendo.

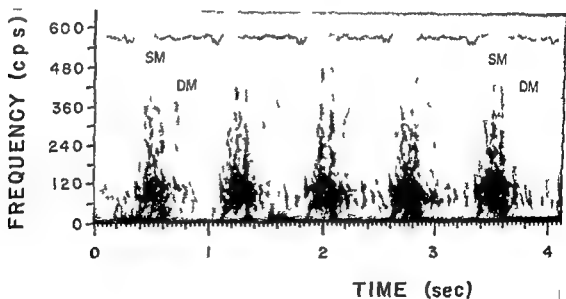


FIG. 2a6 Demonstration of minimal aortic regurgitation in presence of aortic stenosis.

Left sternal border in patient with rheumatic aortic stenosis and regurgitation. A diastolic murmur of typical shape is present. An unusual feature is the continuation of the murmur to one component of S<sub>2</sub> which is probably pulmonary. Left bundle branch block is present with paradoxical splitting of S, i.e. the pulmonary component precedes the aortic. The exaggerated aortic stenosis exaggerates the paradoxical splitting. A second feature of note is the unusually faint high pitched nature of the aortic diastolic murmur. The spectrogram can more easily and accurately demonstrate a faint diastolic murmur in the presence of a loud systolic murmur than can the oscillogram (see p. 81).

(484A) They believe that a form of atrophy which may begin in early childhood and be exaggerated by aging dilatation of the ring and increased mitral valve pressure is responsible for aortic regurgitation with systemic arterial hypertension and the Graham Steell murmur of pulmonary hypertension may frequently have sensation of emulsion passing the mitral valve.

The question of whether aortic regurgitation can occur on the basis of myocardial weakness during heart failure has been discussed on page 183.

Acute dissecting aneurysm may cause aortic regurgitation through distortion of the aortic ring by the medial hematoma (p. 422) but often the mechanism of the association between aortic regurgitation and dissecting aneurysm resides in the fact that the two have a common basis—idiopathic cystic medial necrosis—the Marfan syndrome-hypertension.

In cases of aortic regurgitation one is likely to see dilatotic pockets or swallow spaces directed toward the aorta and located on the interatrial septum a short distance below the aortic valve. Sometimes called in the older literature Zahn's (1601) or Schmincke's (1362) pockets, they represent a cuffing up of the endocardium by the regurgitant stream. (Rarer are pockets facing downward in association with aortic stenosis and pockets in the left atrium facing the mitral valve in mitral regurgitation.) Particularly in cases with an Austin Flint murmur, suspect of the regurgitant stream on the aortic leaflet of the mitral leaflet may produce a perforation there (481). A jet lesion may develop on the intima of the ascending aorta in aortic regurgitation merely because of the large stroke volume and sharp concentrated forceful ejection by the ventricle. It is important to recall that syphilis does not cause aortic stenosis in the actual organic sense.

PHYSIOLOGIC CONSIDERATIONS (427A) Obviously the regurgitation of blood into the left ventricle results in an increase in left ventricular stroke volume since under conditions of adequate function something approaching a normal quota of blood plus that volume regurgitated during previous diastole is ejected with each ventricular systole. The velocity of ejection is increased

since there is a greater volume of blood to be ejected in roughly the same period of time and since the pressure into which the ventricle is forcing is low at the beginning of ventricular ejection.

The collapsing pulse and bounding head of aortic regurgitation are principally a function of the shape of the arterial pulse pressure curve (434). There is what Wiggers terms "a systolic collapse" of the arterial pulse. One wonders whether regurgitation at the aortic valve might not begin before the second sound—it does before the pulmonary component of the second sound. The late systolic element of the mitral murmur shown in Figure 97B might be evidence for this.

Lowenberg (966) refers to cases in which an aortic diastolic murmur present during a period of heart failure disappeared when compensation was re-established assuming that this was in deed aortic regurgitation and not pulmonary

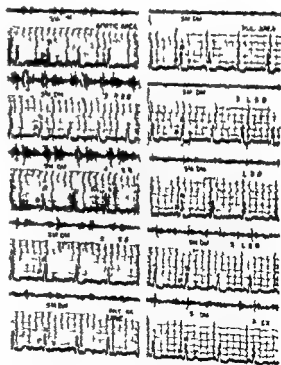


FIG. 25. Aortic aneurysm of aorta of Valvula in a 48 year old woman. The murmurs of aortic regurgitation were best heard along the right sternal border (RSB) than along the left (LSB). A systolic murmur (SM) diastolic murmur (DM) of Harvey (66) and Car

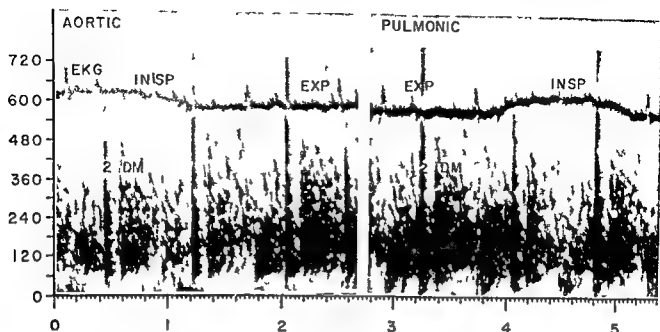


FIG 258 Aortic regurgitation with gap separating  $S_2$  and onset of diastolic murmur

Aortic and pulmonic are in V<sub>1</sub> (413145) 21 year old female patient with rheumatic valvular heart disease predominantly aortic regurgitation and mitral regurgitation. Although not entirely evident the gap between the second sound and the diastolic murmur is striking. Timing with the electrocardiogram and general appearance make it clear that the sound marked 2 is indeed  $S_2$  and not a late systolic click which could create a false impression of gap.

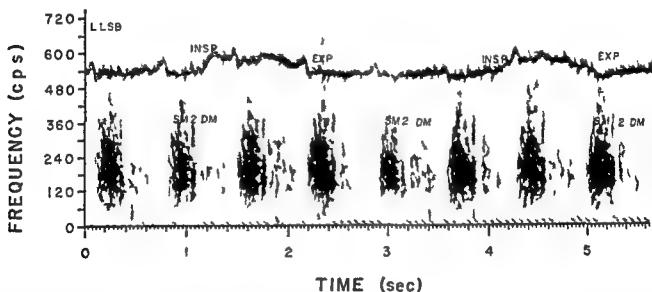


FIG 259 Gap between  $S_2$  and murmur of aortic regurgitation (LLSB) in C (74135a) who had left bundle branch block. The combination of left bundle branch block and aortic stenosis causes paradoxical splitting of  $S_2$ . The Christmassetree murmur of aortic stenosis extends to the pulmonary closure sound. The diastolic murmur begins only after a brief gap. The aortic closure sound is poorly demonstrated.

regurgitation, this "functional aortic insufficiency" could be so identified by comparison of the blood pressures in the arms and legs. Normally there is little discrepancy in the pressures nor is there much in cases of functional insuffi-

ciency even though the diastolic pressure is depressed. In organic regurgitation systolic pressure was 50 to 100 mm Hg greater in the legs. I suspect this differential is spurious. The difference between the two types of regurgitation

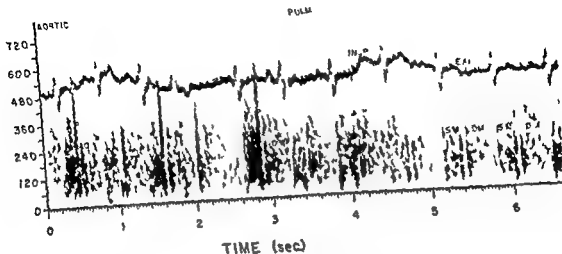


FIG. 70 Gap between  $S_2$  and murmur of A.

Aortic (A) and pulmonary (P) pressure in J. S. (GNSO) 21-year-old female said to have long-standing murmur from birth. ECG showed marked left ventricular hypertrophy. This is probably parallel vessel splitting at decreasing ventricular pressure.

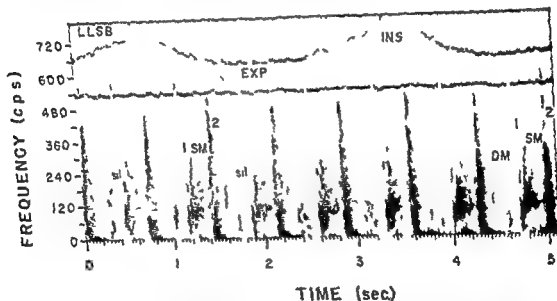


FIG. 71 Interruption of diastolic murmur by atrial systole.

A. C. (GNSO) aut. 4) died at the age of 44 years with the anatomical diagnosis of calcific aortic and mitral stenosis. Regurgitation at these valves was thought to be pre-natal. *SYCG (LLSB)*: A systolic murmur is introduced by an early aortic click. Pulmonary closure is very loud and is preceded by a faint aortic closure sound. A murmur begins immediately with the second sound and ends with a transient which may be either atrial sound or closure sound. There was a normal rhythm and normal ECG interval in this case because of wrong (p. 43) the electrocardiogram does not show the P waves clearly and shows an artificial wave that suggests a wave. Probably atrial systole increases ventricular pressure to the point that there is no longer sufficient aortic ventricular pressure gradient to maintain the murmur.

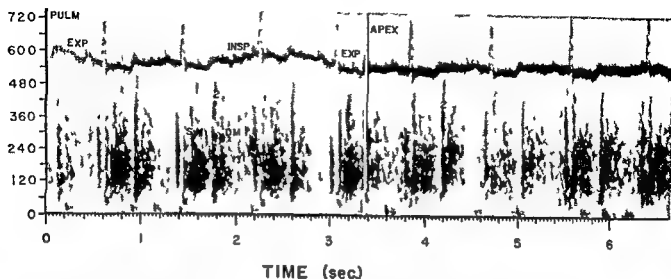


FIG 262 Intersystolic gallop and Austin Flint murmur

Apex (1) and pulmonary area (B) in O. B. (751996) with syphilitic aortic regurgitation. There is in addition to the early diastolic murmur a systolic murmur probably of relative aortic stenosis, a striking atrial gallop and an Austin Flint murmur at the apex.

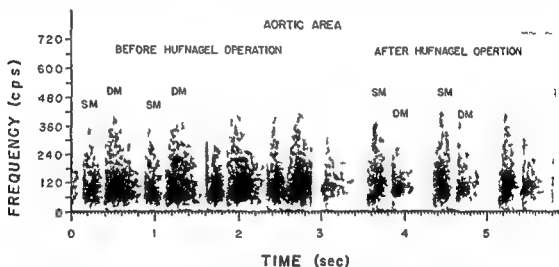


FIG 263 Effect of Hufnagel operation

The aortic diastolic murmur of aortic regurgitation (rheumatic) before and after Hufnagel operation in B. C. (474071). Intensity calibrated SPCG's recorded and analyzed in an identical fashion. The systolic murmur is little changed.

probably is caused entirely by differences in volume of regurgitation—or it may have been pulmonary regurgitation that was present in the group thought to have relative aortic insufficiency.

Left heart catheterization in patients with aortic regurgitation (1116) and observations in animals with an experimentally established model of aortic regurgitation (1347) indicate that in some cases of very large regurgitation the end diastolic pressure in the left ventricle may exceed the pressure measured in the left atrium at

any time in the cardiac cycle. Obviously this observation has several important implications for cardiovascular sound. (1) It is conceivable that the mitral valve will close before the end of diastole and before ventricular systole. This could be a silent process if it occurred slowly, as one would expect to be the case. (2) In those cases in which the valve is already closed at the onset of ventricular systole the first heart sound would be expected to be muffled. (3) Cases would be anticipated in which an intermediate grade of elevation of left ventricular end diastolic pres-

ure exists and pressure in the left atrium with atrial systole may temporarily exceed that in the ventricle. The result might be expected to be the movement of blood through a narrowed mitral orifice with the production of a presystolic murmur--? the Austin Flint murmur.

Regurgitant flow rarely exceed forward flow. The combination of mitral regurgitation with aortic regurgitation results in a decrease in the effective ventricular output according to the studies of Sarnoff and colleagues (171).

**CARDIOVASCULAR SOCIETY** It is proper to peak of the murmurs of aortic regurgitation, because as will be noted the systolic murmur is as much an integral expression of the pathologic physiology of this lesion as the diastolic murmur and the Austin Flint murmur occurs frequently. However the characteristic and pathognomonic murmur is the arterial diastolic murmur previously described in detail (see p. 197).

The location of maximum audibility of the pathognomonic murmur is usually Erb's point but occasionally may be down the right sternal border at the xiphoid, at the cardiac apex or even in the left axilla. It is not yet clear how much significance in terms of etiology of the aortic regurgitation or precise anatomy of the valve lesion can be assigned to the radiation of the murmur. Some have claimed that problems

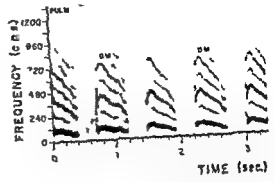


Fig 25a. Retracted aortic ejection from a patient with T. a. (1931-32) 50 year old male. Of note are the following features: (1) an unusually large number of harmonics are present (pulmonary area); (2) there is at the time of atrial systole an increase in the downward slope of the harmonics (cf pulmonary area) at the apex there is an Austin Flint murmur which is partially masked. See Fig. 25B.

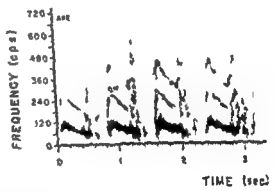


Fig 25b. See Fig. 25a.

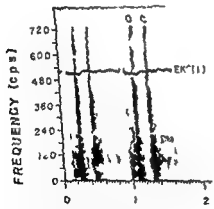


Fig 26. After Hufnagel operation (recor'd) after Hufnagel operation in the same patient as in Figure 25. From area where the sound of the artificial valve is loudest. Note delay between beginning of QRS and valve opening sound (m) on the other hand the valve closure sound (c) almost coincides with S<sub>2</sub> valve beyond the valve is abbreviated.

most radiation down the right sternal border occurs more commonly in aortic regurgitation (44) and (46) the Murmur syndrome coronary insufficiency in (Fig. 23b) and the closing murmur of the aorta (1.00) than in the aortic regurgitation. Aortic regurgitation valve lesion in aortic regurgitation endocarditis may result in a murmur down the right sternal border. The practice of auscultating down the right sternal border should be cultivated. Linn and Quinard (1926) speak of the rare case in which an aortic diastolic murmur is heard only in the first right inter-space. It is worth while emphasizing that aortic diastolic murmurs of more than minimal intensity are transmitted to the apex rather as a rule. Quite aside from the Austin Flint murmur there is opportunity for

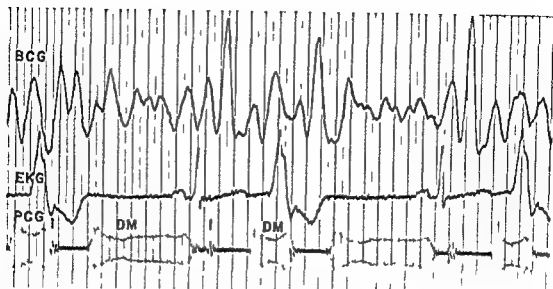


FIG. 266 Retroverted aortic cusp

Oscillogram in patient M. G. (124569). Only the fundamental is demonstrated. The diastolic murmur shows interesting changes in intensity. At its onset it takes a brief time to attain maximum intensity and is decrescendo thereafter until mid diastole when there is a second increase in intensity. Ventricular filling probably produces the retroverted cusp into the regurgitant stream. A third rise in intensity occurs with atrial systole—probably for similar reasons—and is followed by a sharp decline in intensity probably because of abrupt decrease in the aorto-ventricular pressure differential. There is bigeminy caused by digitalis intoxication and with each extra systole a sharp increase in intensity of the diastolic murmur occurs before it is abolished again probably because of displacement of the retroverted element into the regurgitant stream. Waning and waxing of the musical aortic diastolic murmur was noted by Celfand and Bellet (537, Fig. 6) and attributed to the retroversion of two aortic cusps.

Although autopsy examination is not available to complete the evidence this patient was studied with such care over a period of 20 years that the evidence for non-luetic retroversion, tear or perforation of an aortic cusp is convincing. As in this patient, abrupt onset of awareness by the patient of a musical sound in the chest occurred in a number of the patients with retroversion of an aortic cusp on the basis of aphid.

M. G. (J. H. H. 124569), a Negro female, was 54 years old when her sixth admission to the hospital was prompted by the development of a purring sound in her chest four days previously.

She had been well most of her life and previous hospital admissions were for obstetrical deliveries. Repeated serologic tests for syphilis were always negative; there were never previous manifestations interpretable as syphilitic in etiology, and no therapy for syphilis had ever been given.

In 1938 (at the age of 40 years) during her last pregnancy, the blood pressure was 160/110 mm. Hg and cardiomegaly with signs of mild congestive heart failure was present. Tubal ligation was performed. Two years before final admission chest x-ray revealed slight enlargement to the left and there was left axis deviation by electrocardiogram. The blood pressure on repeated examinations during this period was always normal or only slightly elevated. No diastolic murmur was heard.

One week before admission she was awakened with a sensation of fullness in the upper sternal area accompanied by nausea. These symptoms passed off quickly and the patient felt well until four days before admission when while sitting and reading the paper after supper she suddenly became aware of a purring and thumping in her chest. There were no other symptoms.

On examination the blood pressure was found to be 120/65 mm. Hg in both arms. The patient was moderately obese. She was perfectly comfortable. A prominent diastolic thrill was palpable over the entire precordium. The heart was enlarged to the left. Auscultation was dominated by a very loud, cooing murmur which was maximal at the left sternal border but which also was audible over the neck and axilla.

Electrocardiogram revealed the pattern of left axis deviation and left ventricular strain. By x-ray the aorta was dilated and tortuous and the innominate artery was linked and dilated. Serologic tests for syphilis including a treponemal immobilization test were negative. The temperature was always normal and four blood cultures were sterile.

Although no signs of congestive failure were present, prophylactic digitalization was performed. However because of the appearance of numerous ventricular extrasystoles digitalis was discontinued.

The patient did not return for further observation. She had several admissions to another hospital for treatment of congestive heart failure. She died at home 21 months after her first hospital admission.

The mechanism in this patient may have been cystic medial necrosis as discussed in the text. Also as discussed elsewhere arteriosclerosis is possible but less likely cause. Fibrillation (Fig. 253) is yet another

## VALVULAR HEART DISEASE

confusion with mitral regurgitation because of the aortic murmur which in transmission requires a more rumbling quality (p 149). The fact that it begins immediately with the second sound will help identify it. In rare instances an aortic diastolic murmur is louder than even heard only at the apex or in the axilla (the Cole-Cecil murmur).

The shape of the murmur is usually decrescendo beginning essentially with the second heart sound. Occasionally, especially when musical, it may be decrescendo in contour the decrescendo limb being short but steep.

Occasionally on auscultation there appears to be a gap between the second sound and the beginning of the murmur (Figs 238, 239 and 240). The explanation (860) in some cases can be shown to reside in the brief decrescendo which precedes attainment of maximum intensity. In other instances there appears to be a true gap (33b, Fig 31, also 1520) in some cases in which AS and left bundle branch block is present (Fig 239) this may be due to paradoxical splitting of the second sound with delayed appearance of the aortic closure sound.

A long diastolic murmur may continue straight

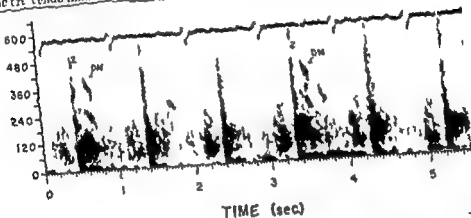


Fig 238 Highly variable musicality in aortic diastolic murmur of patient with aortic regurgitation (JH 7674). At times there seemed to be a relation to respiration, the musical element being greater if expiration.

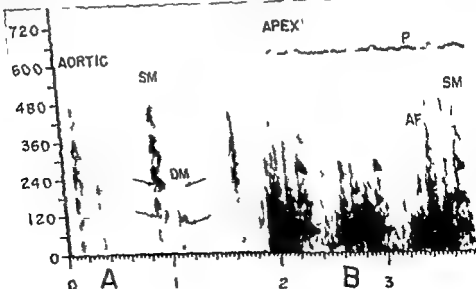


Fig 239 Musical aortic and diastolic murmurs in patient with aortic regurgitation.

JH (7680) age 59. The vibrating element may project into the aorta in axilla. At the apex the vibrating element is below and there is both an Austin Flint murmur and a presystolic gallop.



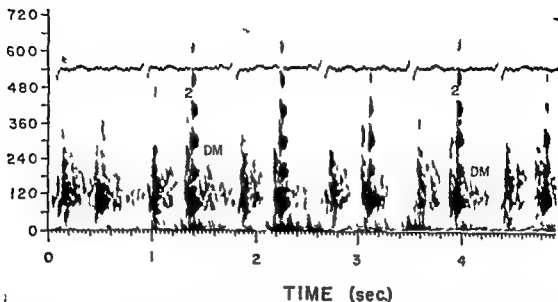


Fig. 269. Circumscribed early diastolic musical element in longer noisy diastolic murmur of aortic regurgitation. Syphilitic aortitis in F. D. (616706) colored female age 60 years at time of this recording. The patient's clinical course has been surprisingly benign.

to the next first heart sound. Occasionally the murmur cuts off abruptly at the time of atrial contraction. Presumably atrial contraction raises intraventricular pressure abruptly to a level such that the pressure differential between aorta and pulmonary artery is below the threshold for murmur production. More rarely when the regurgitant murmur stops abruptly in mid diastole at a point unrelated to atrial systole there is at its end a sound, i.e., a transient which I have termed a riccio sound. These features of the shape of the murmur of aortic regurgitation are displayed in Figure 9b.

The quality of the murmur is usually whiffling, whurring or blowing, depending to some extent on how loud it is. On transmission to the apex the quality of the murmur is likely to be altered as a result of selective transmission of those components at frequencies in the range of the natural frequency of the thorax. This tends to be at the lower end of the frequency scale. The fact that the murmur is lower pitched and more rumbling at the apex does not necessarily imply an origin different from that of the murmur heard at the base.

Occasionally the murmur is musical. Retroversion of a cusp (Figs. 264 to 268) with creation of a lip which is free to vibrate in the regurgitant stream is the classical anatomic basis but fenestration and other less easily described deformities

of the cusp may produce a musical murmur. Occasionally in so-called calcific aortic stenosis there is a musical diastolic murmur as well as the systolic one. In such cases the deformed valve orifice functions as the generator of a musical murmur with flow in both the forward and backward direction.

The musical murmur with fenestration of a cusp/bacterial endocarditis and valve deformities other than retroverted cusp is rarely as pure as that associated with the latter lesion. It usually is a mitter of some harmonics in the midst of a noisy murmur. Occasionally the murmur of retroverted cusp may disappear being replaced by a conventional noisy aortic diastolic murmur. This I have observed at least twice (P. C. 424089, I. S. 103485). In one case a patient with syphilitic aortic regurgitation (F. D. 616706) the musical murmur was very short and limited to the first part of diastole. Two years later it had become a more conventional long musical diastolic murmur of retroverted cusp.

Usually in both rheumatic fever and in syphilis once an aortic diastolic murmur is heard in indubitable form it persists thereafter. There are reported exceptions, however, in both diseases (739).

The systolic murmur of aortic regurgitation is

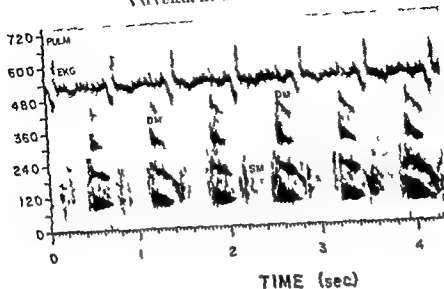


FIG. 20 Same patient as in Fig. 19, 10 years later. The mitral murmur now occupies most of this cycle. Pulmonary area. EKG = inverted limb lead I?

the result of the large stroke volume and the high velocity of ventricular ejection is outlined under Pathologic Considerations. Distention of the ascending aorta, a likely accompaniment of aortic regurgitation on the basis of syphilis or of the Marfan syndrome contribute to the systolic murmur. On rare occasions the systolic murmur may be so intense that a thrill is felt. Leatham (1962) observed three such patients with aortic regurgitation due to syphilis. The differentiation of relative aortic stenosis from true organic stenosis is frequently difficult on the basis of the sound alone. The later the frequency intensifies peak of the murmur the more likely is there to be organic stenosis.

The *blubbery* Flint murmur of aortic regurgitation is a diastolic murmur at the apex with an early systolic character, sometimes resembling those of the murmur of mitral stenosis (Fig. 2b2). It is difficult to tell from Flint's original report (1941) whether the murmur which he characterized as blubbery was pre-systolic or mid-diastolic. However, in another publication 24 years later he leaves no doubt that it is a mid-diastolic pre-systolic murmur. It seems that either a mid-diastolic or a pre-systolic typical murmur may be associated with pure aortic regurgitation. I would re-emphasize on why the Flint eponym should not be applied to both. The change in quality of the primary murmur of aortic regurgitation on transmission to the apex should not occasion confusion with

the mid-diastolic Flint murmur. Also not to be confused are a pre-systolic gallop or a crepant  $S_2$  which may occur with aortic regurgitation (1977).

When tachycardia is present the erroneous diagnosis of mitral stenosis is especially likely because a diastolic murmur may occur at a time when the factors are ripe for a mid-diastolic Flint murmur to operate. Furthermore as with any tachycardia the first sound becomes accentuated while the ringing first sound of MS. An aortic ejection sound occurs commonly in aortic regurgitation and tends to be well transmitted to the apex. The preceding first sound and/or a pre-systolic Flint murmur may with the ejection sound suggest MS. In the oscillogram too the late ejection sound would be consistent with the delayed  $S_2$  of MS. In the spectrogram the characteristic features of clicks and snaps (p. 175) are likely to permit correct identification of the ejection sound.

A number of theories have been proposed for the genesis of the Flint murmur (120). Distention of the ventricle probably contributes as in some cases of relative mitral stenosis. On other bases Currens and co-workers (120) stated that left ventricular distention is apparently not essential for the production of the Flint murmur but may predispose the heart to such a murmur. Actually I doubt that a Flint murmur ever occurs without at least slight ventricular distention. Any ex-

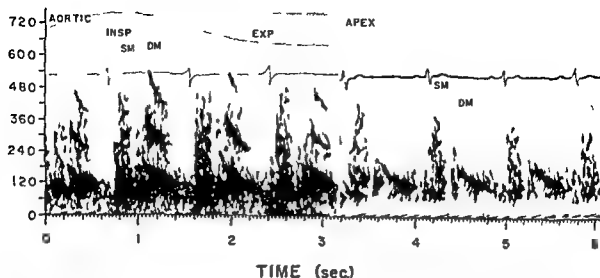


FIG 271 Preferential transmission to apex

Recorded in H I (765151) with musical aortic diastolic murmur audible as far as each olecranon and the lower abdomen and back. The intensity and frequency level of the murmur was accentuated in early expiration. There is a change in tonality on transmission to the apex caused by preferential transmission of the fundamental.

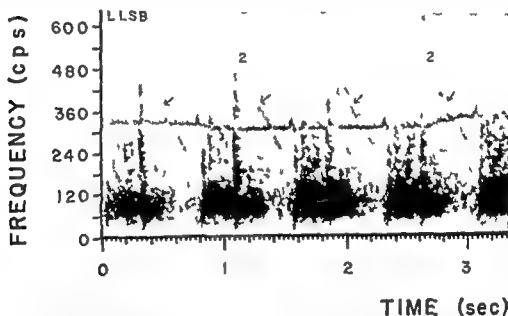


FIG 272 Unusually high pitched musical diastolic murmur in syphilitic disease of aortic valve

LSB in N G (720022) 50 years old with historical and serologic evidences of syphilis. The musical element of his diastolic murmur is unusual for syphilis in that it consists of a single harmonic and has an unusually high pitch exceeding 400 cps at its peak. The harmonic has the typical crescendo-decrescendo chevron pattern. An early systolic click of dilated aorta introduces a decrescendo systolic murmur.

ation of ventricular dilatation is notoriously unreliable; the stroke volume is necessarily increased in aortic regurgitation and presumably diastolic volume of the ventricle is also increased. Increased flow across the mitral valve may be a contributory factor, since relative mitral insufficiency on the basis of ventricular dilatation is to be expected. Filling of the ventricle from two

sources may result in the mitral cusps' attaining a more nearly closed position at the end of diastole. An important factor may be more direct impingement of the regurgitant stream from the aorta on the anterior aortic leaflet of the mitral valve with displacement of the cusp into the stream of blood entering the ventricle from the left atrium. This would be expected to produce not only a

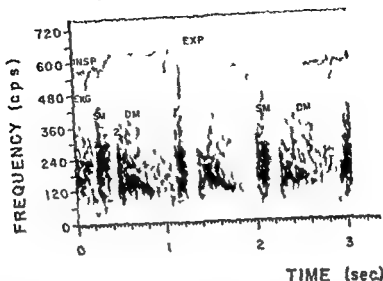


Fig. 73. Murmur diastolic murmur with calcific aortic valve disease.

Aortic area in A. F. (3333) 73-year-old female who in childhood had growing pains but no definite rheumatic fever. Dyspnea and ankle edema brought her to the hospital. Examination revealed a blood pressure of 210/0/0 mm Hg, kinked carotid on the right, a loud systolic and diastolic murmur both rather musical to the ear and left ventricular enlargement by x-ray and ECG. It was concluded that she suffered from calcific aortic valve disease which was probably atherosclerotic on a rheumatic basis. The musical elements of the diastolic murmur are well shown and those of the systolic somewhat less well shown.

pre systolic but also at times a murmur beginning earlier in diastole following the second sound by a brief gap. The theory just mentioned has been supported by most recent writers (e.g. 320) and was proposed by Dickinson (322) in 1908. On the basis of ten cases Gault (381) concluded that there is a characteristic deformity of the right anterior coronary cusp associated with Austin Flint murmur—a concave cup-shaped deformity at the inner portion of the leaflet situated to direct or groove the regurgitant blood toward the lower portion of the anterior mitral curtain. The latter exhibited on its ventricular aspect a variable degree of endocardial thickening which was interpreted as fractional sclerosis.

The second sound in aortic regurgitation is probably accentuated in a majority of cases. In mild rheumatic aortic regurgitation fibrosis of the valve probably is possible and the same factor probably operates in more severe cases. In a phibitic aorta the ringing second sound is well recognized. Another contributing factor in the accentuated aortic second sound may be the

systolic collapse phenomenon. More rapid fall in aortic pressure with accelerated aortic closure and accentuation of  $A_2$  may occur.

The peripheral auscultatory signs of aortic regurgitation are often striking but rarely of specific diagnostic usefulness over and above that of the blood pressure determination and the primary or central auscultatory signs. The main findings of auscultation over peripheral arteries are pistol shot sound, Duroziez sign and Traube sign. These have been described earlier (p. 234). The pistol shot sound is a single transient related to the water hammer pulse, the Duroziez sign is a double murmur elicited by compression of a peripheral artery with the margin of the bell of the stethoscope, the Traube sign consists of two sounds heard without compression of the artery.

#### MITRAL STENOSIS (MS)

ETIOLOGIC AND PATHOLOGIC CONSIDERATIONS (133, 132, 134). Rheumatism is virtually the sole cause of true or organic mitral stenosis of significant proportion. Congenital mitral stenosis (p. 38) has been recognized with increasing

frequency in recent years, but is still a rarity (456) Trauma was thought to be the cause in one patient (50), who the authors thought had sustained a hematoma of the aortic leaflet of the mitral valve

Rheumatic fever causes gluing together of the mitral cusps, which themselves become thickened by a fibrous and sometimes calcifying process. The same scarring process involves the chordae tendineae, which may become shortened, if not actually shortened (583), their effective length is reduced by the fact that in the scarring process they become glued together at their cuspal ends and, in effect, incorporated into the cusp. Most mitral orifices sufficiently stenosed to give symptoms for which valvulotomy is indicated have a cross sectional area of the order of one square centimeter or less. (The normal effective mitral orifice area is of the order of 5.0 cm<sup>2</sup>) The end stage is appropriately referred to as a 'fish mouth' valve. The aortic leaflet of the mitral valve usually retains enough pliability to play a role in the mitral opening snap and probably in the snapping quality of the first heart sound. Extensive calcification is usually associated with loss of the pliability of the aortic leaflet and with corresponding changes in the auscultatory findings.

PHYSIOLOGIC CONSIDERATIONS (576, 579) Physiologic factors are intimately related to the changes in cardiovascular sound in mitral stenosis. For example, the level of left atrial pressure or rather the atrioventricular pressure gradient is related to the degree of delay of  $M_1$  and the interval between S and the opening snap, the volume of blood flow as well as the pressure gradient is related to the intensity of the diastolic murmur. However these features can be best discussed in relation to the auscultatory changes.

Adopting a method commonly employed by hydraulics engineers Gorlin and Gorlin (575) proposed the following formula for estimating effective diastolic mitral valve area ( $MVA$ )<sup>2</sup>

$$MVA \text{ (in cm)} = \frac{MVF}{31 \sqrt{PC - C}}$$

where  $MVF$  is mitral valve flow (cardiac output per minute of diastolic time) and  $PC$  is pulmo-

nary capillary ("wedge") pressure. Both of the data can be derived from right heart catheterization. The reader will recognize that the above formula is a special case of the following relationship: flow is proportional to orifice area and to the square root of the pressure gradient (differential pressure)

$$MVF = I \times MVA \times \sqrt{\text{left atrial pressure} - \text{left ventricular pressure}}$$

The constant "I" was determined to be 31. Pulmonary capillary pressure is a measure of left atrial pressure. Mean diastolic pressure in the left ventricle is assumed to be 5 mm Hg. Normally the effective mitral valve area measures approximately 5 cm<sup>2</sup> as calculated from physiologic data applied to the above formula and as checked anatomically.

CARDIOVASCULAR SOUND I tell medical students that when they have mastered the auscultatory phenomena of mitral stenosis in all its ramifications they have mastered the entirety of clinical auscultation of the heart. This relatively complex subject can be discussed under the following headings:

- (1) The timing and quality of the first heart sound
- (2) The systolic murmur
- (3) The second sound
- (4) The mitral opening snap
- (5) Estimation of the severity of mitral stenosis
- (6) The third heart sound
- (7) The passive diastolic murmur
- (8) The atri-systolic murmur
- (9) The pulmonary early systolic click
- (10) The Carotid Steel murmur
- (11) Post valvotomy changes
- (12) Conditions precluding auscultatory signs simulating those of mitral stenosis

The first heart sound (mitral closure sound) is delayed and ringing in mitral stenosis. The degree of delay bears a direct and rather quantitative relationship to the level of left atrial pressure and therefore to the severity of the mitral obstruction (793, Fig. 3). The Q1 interval in adults is normally between 0.02 and 0.06 sec. I could not find (875) found an average figure of 0.05 sec. In individuals with mitral stenosis the interval

<sup>2</sup> As indicated in the footnote on page 263 the Gorlin formula applies only to small or malshaped orifices not to the normal orifice.

may be as long as 0.12 sec. The pressure in the left atrium is elevated often to values in the range of 3 to 40 mm. Hg in severe cases of mitral stenosis. The mitral closure sound does not occur until pressure in the left ventricle exceeds that in the left atrium. This may take as long as 0.07 sec. after the onset of left ventricular contraction. It will be noted that there are two components to the Q1 lag—one electrical if you will—the delay between the beginning of the QRS and the onset of ventricular contraction as indicated by the onset of pressure rise—the other mechanical—the delay between the onset of contraction and the closure of the mitral valve when pressure in the left ventricle exceeds that in the atrium. It is the latter which is prolonged in these cases.

Bundle branch block—by prolonging the first phase of the lag—might be expected to confuse the picture and vitiate the usefulness of the Q1 measurement in gauging the severity of the mitral stenosis. However the evidence of Braunwald indicates no delay in the onset of contraction in either ventricle in most cases of bundle branch block (p. 166).

It has recently been demonstrated (87a) that a prolongation of the Q1 interval occurs in association with a tension arterial hypertension (see p. 447). Hypertension can therefore interfere with phonocardiographic estimation of mitral stenosis in two ways: the manner just mentioned and the effect on the interval between the second sound and the opening snap (see below).

In the oscillogram it is the beginning of the first rapid vibration which are used in making the Q1 measurement. These are almost always, if not the first large vibration. There are often some earlier vibrations of small amplitude preceding the mitral closure sound. These were thought to be related in some manner to atrial systole until it was noted (308) that they persist in cases of atrial fibrillation. They may represent the elusive and much debated muscular (i.e. myocardial) contribution to the first heart sound. It is not surprising that they precede the valve closure sound occasionally especially in the spectral phonocardiogram one sees a discrete sound just preceding the sharp mitral closure sound which satisfies the criteria for a tricuspid closure sound (Fig. 281). That this is not more often seen in mitral stenosis is probably related to the fact

that normally mitral closure slightly precedes tricuspid closure. Considerable delay of the mitral closure sound is necessary before a paroxysm of the two becomes evident through a paradoxical sequence of closure. Hultgren (72b) has demonstrated a sound preceding the mitral closure sound and having characteristics one might predict for a tricuspid closure sound. Specifically its intensity varied inversely with the duration of the preceding diastole in cases of atrial fibrillation. The sound was still present when recordings were made from the exposed heart at surgery, impaled against the thoracic cage, not its neck, etc. Some reported examples of so interpreted presystolic murmur persisting after the development of atrial fibrillation (7A, 1388) were probably cases of a tricuspid sound preceding the mitral closure sound.

With extensive calcification of the mitral valve the first sound may be diminished even though tight mitral stenosis is present and is the predominant lesion (furthermore an opening snap may be absent in such cases). The intensified ringing M<sub>1</sub> persists after mitral commissurotomy unless a good deal of mitral regurgitation has been inadvertently produced.

The oscillogram displays the unusually great intensity of the mitral closure sound and the SPCG displays this plus the frequency characteristics responsible for its ringing quality to the ear. The ringing heart sound, to ordinary non-heart sound is a mitral murmur is to noisy murmur. In the SPCG the ringing M<sub>1</sub> of mitral stenosis tends to display harmonic pattern just as do mitral murmurs. Furthermore it has components of a higher frequency than normally and of course its overall intensity is increased.

The great intensity and the particular quality of the first sound in mitral stenosis are the result mainly of the fibrotic change which has taken place in the mitral valve curtain especially the vortice or antero-apical leaflet. Anatomically it is impossible to imagine much sound being produced in coaptation of the margins of the cusps which are relatively close together. Sudden tensing of the shortened stiffened chordae tendineae may contribute. Observations on the first heart sound in mitral stenosis appear to shed light on two important physiologic features of the normal heart sounds and normal valve func-

tion (1) the first sound is related, in the main, to AV valve closure, (2) valve closure sounds are produced more by tensing or snapping of the belly of the valve cusps than by collision of the coapsing valve margins.

Normally with atrial fibrillation alone there is variability of the intensity of  $S_1$  as indicated in Figure 447 (p. 437) at values of diastole between 0.20 and 0.25 sec the intensity reaches a minimum, at still longer diastolic periods there may be a secondary increase in intensity. According to Rivin and Bershof (1247) in severe mitral stenosis there may be essentially no variation in

the intensity of  $S_1$ . In a certain number of patients, intermediate as to severity (this category was represented by four out of ten patients studied) there is a slow decline in intensity reaching a minimum value at diastolic periods of 0.5 to 0.7 sec. The explanation offered for the finding in the normal situation is that as ventricular filling progresses the valve clets "float up" to a semi-closed position and make less noise in closing. In mitral stenosis diastolic filling is delayed and prolonged the normal process of "floating up" takes a longer period or does not occur at all because of elevated left atrial pressure. In Figure 282 is presented a case of mitral stenosis and atrial fibrillation in which  $S_1$  varies in intensity depending on the length of the preceding diastolic period.

A systolic murmur may occur at the apex if there is mitral regurgitation of any degree. It was formerly thought that for practical purposes mitral stenosis never exists in the absence of some degree of mitral regurgitation. That this is not true is one of the many facts revealed in connection with mitral valve surgery. So called "pure" mitral stenosis does exist. Even though the stenotic orifice is rather rigid the cusp curtain may be sufficiently pliant to appose above the orifice (408). The mitral mechanism then becomes essentially a flutter valve. A systolic murmur is sometimes heard at the apex in cases of mitral

### DUROZIEZ'S ONOMATOPOETIC DEVICE

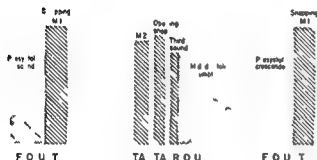


FIG 274 Duroziez's fout in tarou of pure mitral stenosis

The sound marked third sound is usually little more than a concentration of the first part of the mid-diastolic murmur in those cases in which the rumble does not begin immediately with the opening snap.

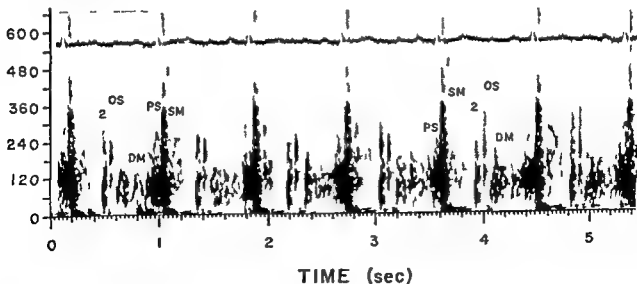


FIG 275 Apex D.C. (687240) with predominant mitral stenosis. Note the close reproduction of the Duroziez device shown in Figure 274.

tensio: yet no regurgitant jet is found at operation assuming that the heart is at the time beating as vigorously as before thoriotomy or at least sufficiently vigorous for regurgitation to be expected—operationally speaking, if the blood pressure is in the usual range—then the basis for the aortic murmur in many of these cases may be relative tricuspid insufficiency caused by dilatation of the right ventricle rather than mitral regurgitation (483 1357 1482). Accentuation of the aortic murmur with inspiration helps in this identification but unfortunately is not always present. The murmur of tricuspid regurgitation is likely to be heard unambiguously far to the left in this situation because of the dilation of the right ventricle and clockwise rotation of the heart.

Mounsey and Bridgen (1126) remarked on the frequent occurrence of a short decrescendo early aortic murmur at the apex limited to the first part of systole and not related in any consistent manner to the finding of mitral regurgitation at operation. This murmur is illustrated in several of the recordings presented here e.g. Figure 273. This murmur unlike the murmur of the usual mitral regurgitation is not holosystolic.

There may be a short not loud early aortic murmur in the pulmonary area and just below. The site of origin is the dilated pulmonary artery and the murmur is often introduced by an early aortic click (see later).

A systolic click in cases of mitral stenosis is often immediately short. Even on incultation the second sound frequently seems to come very soon after the first sound and the principal finding is a long diastolic rumble ending in a sharp first sound. The interval between the two heart sounds is

shortened because of delay in the first heart sound. In addition there probably is a true abbreviation of systole due to the small stroke volume.

The second sound in the pulmonary area in mitral stenosis is usually accentuated as a result of pulmonary arterial hypertension. In pure mitral stenosis the heart sounds in the aortic area especially the second sound are usually strikingly faint especially in comparison with the loud  $P_2$  and ringing  $M_2$ . This is Kocher's sign—loud  $P_2$  faint  $A$ . The faint aortic sound undoubtedly is related in part to the relatively low aortic pressure in cases of pure mitral stenosis but also to clockwise rotation of the heart which occurs in these cases and tends to swing the base of the aorta to the left and closer to the left terminal border. The clockwise rotation of the heart results in considerable part from the enlargement to the right of the left atrium which lies on the posterior aspect of the heart. When  $A$  is loud in combination with sign of mitral stenosis one must suspect the presence of aortic valve change specifically fibrosis. When  $A$  is loud a diastolic murmur at the left terminal border can be expected as representing aortic regurgitation rather than a Graham Steell phenomenon.

Usually at the apex too the second heart sound is faint in mitral stenosis. This is because the aortic closure sound is normally responsible for  $S_2$  at the apex. With pronounced accentuation the pulmonary closure sound may be transmitted to the apex the clockwise rotation of the heart with the result that the right ventricle contributes the apex contributes to this transmission.

Splitting of  $S_2$  does not contravene to a prevalent impression occur to an impressive degree in mitral stenosis. The impression that it does has been created by the fact that the opening snap is more often than not clearly audible in the pulmonary area. True splitting of  $S_2$  is more a feature of mitral regurgitation.

The mitral opening snap ( $OS$ ) is the closest approximation to a unique and pathognomonic auscultatory sign in mitral stenosis. It immediately follows the second heart sound being separated from the aortic closure sound by an interval dependent on the level of atrial pressure and therefore the grade of mitral stenosis. The  $OS$  measurement is then a gauge of the

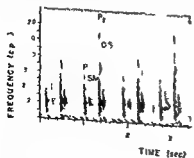


FIG. 276. Pulmonary area: in pure mitral stenosis with mitral rhythm. Opening snap louder here than at apex.



severity of the mitral stenosis, just as is the Q 1 measurement. The S-OS interval may be as little as 0.05 sec. in severe cases of mitral stenosis. In mild cases, especially cases of mild stenosis in association with systemic arterial hypertension (see Fig. 289), the S-OS interval may be as long as 0.14 seconds. After mitral commissurotomy, the opening snap persists more often than not and the S-OS interval lengthens to values of as much

as 0.14 sec. if satisfactory relief of the mitral obstruction has been achieved.

The interval between S and OS varies in atrial fibrillation, depending on the duration of the preceding diastolic period (Fig. 278). With longer diastolic periods there is a longer time for decompression of the atrium and the next OS occurs later with a wider S-OS interval. The converse is true with short diastolic periods. In

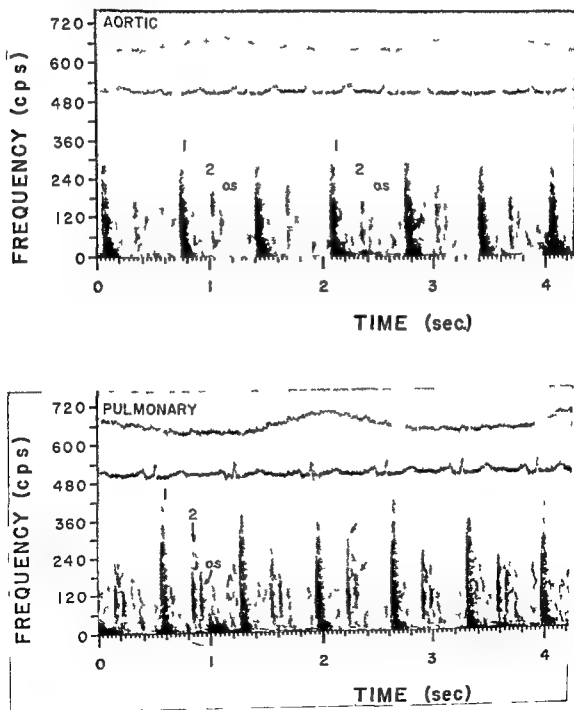


FIG. 277 A (upper) and B (lower). See legend with Figures 277 C and D.

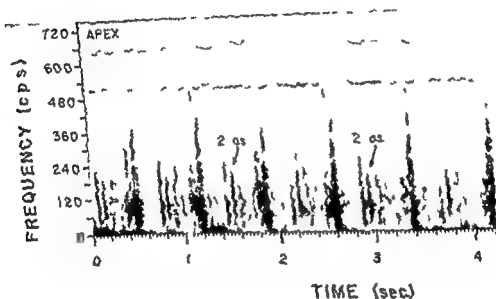
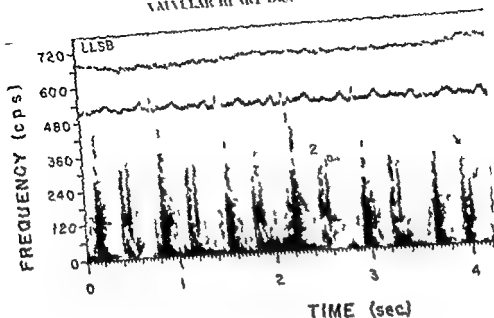


Fig 1. B Typical rheumatic mitral stenosis

In the tracing of Fig 1A the top trace recording in parasternal movement is upward and the parasternal movement is downward as in all other recordings in this group. In it is the area the heart sound are characteristic of a stenotic aortic valve and aortic regurgitation. The heart sound is more typical of aortic regurgitation relative to the QRS indicates that it is the aortic valve and heart in the aortic area with poor clarity because of it accentuation. No splitting of  $S_1$  demonstrated as aortic closure is representative. The opening snap (OS) is well demonstrated in the aortic area in other areas. In the pulmonary area on the left and right second sound is split especially during inspiration (see the upper arrow in the area of the fourth trace in B). The simultaneous demonstration of a split second sound and the opening snap leaves little doubt of the identity of the opening snap. There is probably little pulmonary hypertension in this case. The split second sound and opening snap are similarly well demonstrated at the lower left third border. At the apex the mitral opening snap is accentuated and delayed. The second sound is split and is usually the cause of the result of the valve closure. The aortic valve closure begins immediately with the opening snap and first decrease in intensity in parasternal

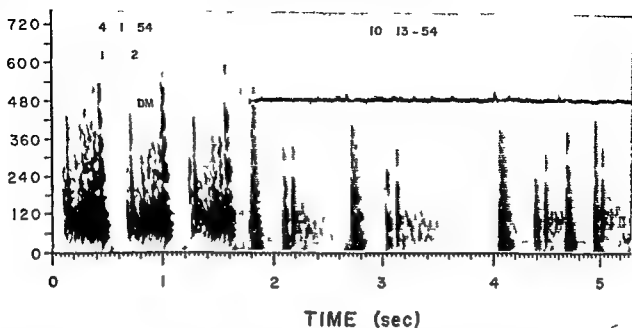


FIG. 278 Effect of atrial fibrillation in mitral stenosis

Apex in S O \ (616116) before and after development of atrial fibrillation. The first recording has a diastolic rumble beginning immediately with the opening snap. With development of fibrillation the presystolic element disappeared. In the first recording the S<sub>2</sub> OS interval is short because of the rapid heart rate and short diastolic periods. In the second record it is longer and varies directly with the length of the preceding diastolic period. Q<sub>1</sub> S<sub>1</sub> delay is also demonstrated.

the upright position the S OS interval is longer than in recumbency (1364, 1454). Mounsey (1124) found no respiratory variation in the interval but Schoelmerich and Gehl (1364) did.

Well (1525-1527) found a rough correlation between size of the mitral orifice as discovered at operation and both the Q<sub>1</sub> interval and the S OS interval. The correlation was much improved by relating Q<sub>1</sub> minus S OS to mitral size. (Both values were corrected to a cycle length of 0.8 sec.) He thought the improved correlation resulted from a cancelling of the effect of varying amounts of fibrosis and calcification of the valve; possibly fibrosis and calcification would increase both the Q<sub>1</sub> and the S OS interval. Also the two measurements together might he thought reflect mean diastolic gradient more accurately. The study of Byer Wolter *et al* (64, 66-158) correlating pulmonary capillary pressure with S OS interval seems to indicate that fibrosis and calcification in fact have no important effect per se—pressure differential is the only factor of importance in determining variation in the intervals in question. (The S OS interval was the one specifically studied by Byers group [see p 190].) Wells' failure to demonstrate a closer

correlation in the first instance might have been the result of the inaccuracies of the measurement of the mitral valve by palpation at surgery. Furthermore taking only valve size into account and not flow—the other factor in determining left atrial pressure—would not be expected to result in perfect correlation. However when correlations with the pressure gradient at operation were attempted he again found that the best correlation was provided by the index of corrected Q<sub>1</sub> minus corrected S OS.

Julian and Davies (761) could find no satisfactory correlation of the Q<sub>1</sub> interval with pressure in the left atrium although the S OS interval correlated well. They suggested that the lack of correlation of the first was because of the relatively wide range of values in normals (0.02 to 0.06 sec., according to Kelly (778)). Difficulties in obtaining a satisfactory correlation of grade of mitral stenosis with the Q<sub>1</sub> and S OS interval may have been due in some instances to uncertainties in the identification of the mitral closure sound and the opening snap in the oscillogram.

Kuo and Schnabel (827) have presented evidence that when hemodynamically significant mitral regurgitation or aortic regurgitation is

present variation in the Q1 and S-O5 interval depending on the length of the preceding diastolic period does not occur.

The opening snap is a name suggesting a dry snapping sound as a rule. However this is not always definite to the ear. Furthermore the opening snap is situated so close to S in cases of severe mitral obstruction that the S-O5 combination in the past has been frequently misinterpreted as a split second sound peculiarly a split I. The confusion is compounded by the fact that contrary to what might on first thought be considered likely the mitral opening snap is well heard—in fact usually better heard than at the apex—at the left sternal border and pulmonary area and even in the aortic area. It can be detected in the upper sternal notch and the right base of the neck in some instances. Linn (1912-1913-1917) has pointed out that whereas the mitral opening snap is usually audible in the upper sternal notch a split second sound is not—a possibly helpful differential point.

In the phonogram there is nothing particularly unusual about the opening snap as compared with other transient. In the S-PCC the opening snap usually has the features which characterize snaps and clicks in general and permit its differentiation from a valve closure sound: (1) brief duration (2) a frequency bottom which does not come to zero (3) a tendency to pure frequency content.

Wood (1958) found that the opening snap was absent in a case of predominant mitral stenosis in which aortic regurgitation was also present. He suggested that the regurgitation might alter the aortic leaflet of the mitral valve during isometric relaxation and early diastole might prevent the sudden blowing of the mitral valve responsible for the snap. Callaghan (1952) in one case and Moore and Dachs (1955) in two failed likewise to detect an opening snap when aortic regurgitation was present. On the other hand Marcolles and Wolferth (1950) found five cases of opening snap in spite of associated aortic regurgitation.

Often the pulmonic diastolic murmur does not

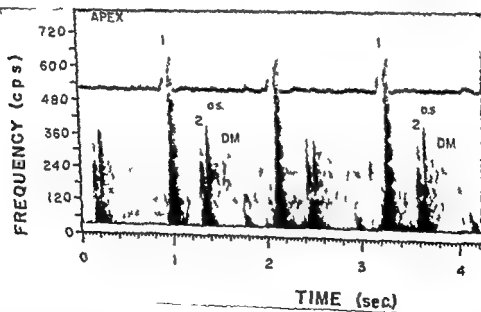


Fig. 29 Rheumatic mitral stenosis with unusually high pitched diastolic murmur

In this recording from the apex the appearance of the characteristic snapping mitral first sound is displaced slightly higher than normal, has a greater frequency span and has a conspicuous harmonic pattern. Thus the mitral closure sound is displaced relative to the QRS as is followed by a striking opening snap. The opening snap is followed immediately by a diastolic murmur of appreciably higher pitch than is usually found in mitral stenosis. All features of the cycle are typical of mitral stenosis of moderate to severe degree. That the murmur in fact had its origin at the site of mitral closure is supported by its disappearance after mitral valvulotomy. In spite of the presence of sinus rhythm no presystolic component of the diastolic murmur is demonstrated in this recording. Electrical interference at 170 cycles and to some extent at 240 cycles is present.

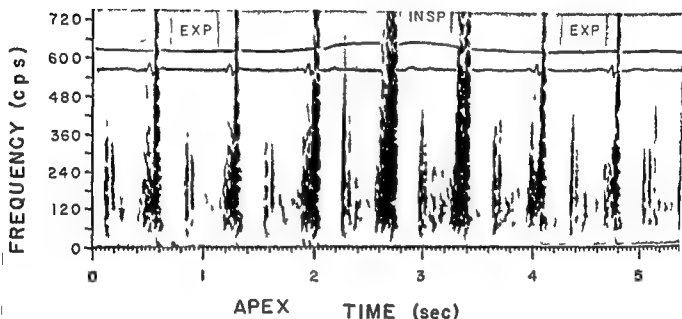
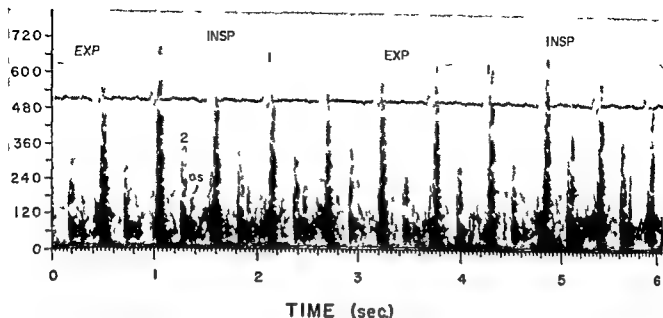


FIG. 29 Changes in the diastolic murmur of mitral stenosis with respiration

(Above) with inspiration the second sound becomes split and it is then the second component (pulmonary valve closure) which dominates. The fact that pulmonary valve closure is so well heard at the apex indicates considerable pulmonary hypertension. The diastolic rumble is intensified early in expiration.

(Below) apex in M. W. (618373) 33 years old and asymptomatic. The presystolic murmur is accentuated in inspiration and early expiration. S<sub>2</sub> OS interval is shortest during cycles with loudest diastolic murmur. All clinical and radiologic evidences point to mitral stenosis not tricuspid stenosis in this patient.

begin immediately with the opening snap but only after a brief interval. In some of such cases there is at the beginning of the murmur a concentration of vibrations which should be considered a sound. Since it has the temporal relationship of a third heart sound, I will so refer to it. It is difficult to

imagine there being a rapid filling sound arising in the left ventricle in MS in view of the impediment to ventricular filling. It may be a third sound produced in the right ventricle (1588 1062). Contro (285) found a third heart sound in 5 of 84 cases of presumed mitral stenosis.

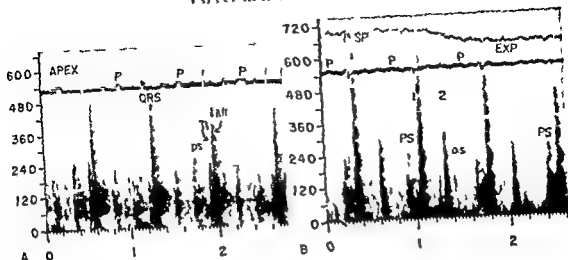


FIG 281 Rheumatic mitral stenosis with prolonged atrioventricular conduction

Fundamentally the presystolic murmur of mitral stenosis is the same as the systolic murmur of aortic or pulmonary stenosis. All are ejection stenosis murmurs. In the case of the presystolic murmur of mitral stenosis the typical Christmas tree pattern is cut short by the snapping first sound in the atrioventricular interval of normal duration. When the atrioventricular interval is prolonged as in the case of the typical pattern becomes evident (left) the first sound is split. Tricuspid closure occurs in its normal relation to the QRS. Mitral valve closure which normally occurs slightly earlier than or coincident with tricuspid closure is delayed and accentuated. Finally in mitral stenosis the presystolic murmur of course the tricuspid closure sound which becomes evident only in cases such as this or case of atrial fibrillation (right) the same Christmas tree pattern of the presystolic murmur is demonstrated because of RR prolongation. The recording was made after mitral valvulotomy. A faint opening snap persists.

One of the five in fact had pure mitral regurgitation. The other four had tricuspid regurgitation in association with mitral stenosis. Contrary conclusion that the right ventricle was the site of origin of the third sound.

What the difference is between cases in which the early diastolic murmur begins immediately with the opening snap and those in which it begins only after a brief interval is not clear. Although there might be an anatomical basis the difference more likely is quantitative than in severe cases that the murmur begins with the opening snap.

The presystolic diastolic murmur is rumbling in quality. With long diastolic period it is likely to display a somewhat decrescendo character. It is the only part of the diastolic murmur which persists when atrial fibrillation supervenes as a variation on the usual rumbling quality. White (1937) p. 97) writes: "Rarely the murmur of mitral stenosis may be a gentle and moderately high pitched flow. We have observed two or three such patients (see pp. 274 and 282). In one the murmur had a rather scratchy quality. The

intensity and duration of the murmur are more reliable indices of the severity of mitral stenosis (10,12).

The presystolic or better atriosystolic murmur

In the 1900s Raymond Tripp and Dixie and also Brackbill (see p. 21) raised the question of whether the so-called presystolic murmur may not really be presystolic. The question was reported by Callaway (in 1910) and by Nichols and colleagues (1947). Callaway was puzzled by the fact that they did not find as they would expect a gap even a millimetre between the atriostolic murmur and the Nichols group thought that at surgery the polythene finger in the left atrium affected the valvular mechanism of the mitral valve. It followed toward the atrium with early ventricular contraction and that the valvular mechanism led to the so-called presystolic murmur which would be more accurately called protosystolic. Callaway (in 1910) suggested early mitral regurgitation to account for the obliteration of the gap between the atriostolic murmur and the snapping S.

It is true that with a RR interval of normal length the presystolic murmur continues into electrical systole of the ventricle so it continues until well after the onset of the QRS the delay in S increases the early systolic extension of the murmur. Both the Nichols and

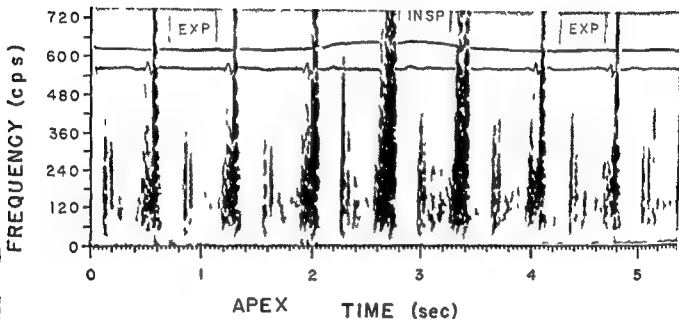
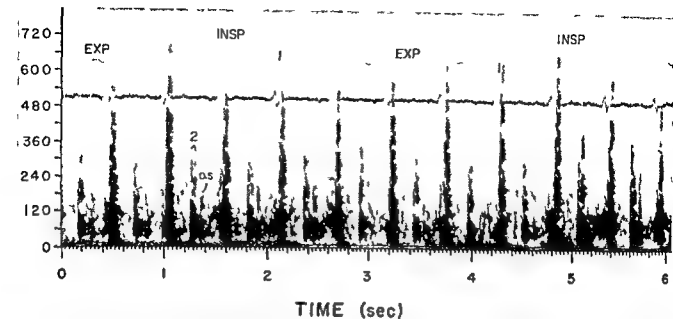


FIG. 280 Changes in the diastolic murmur of mitral stenosis with respiration

(Above) with inspiration the second sound becomes split and it is then the second component (pulmonary valve closure) which dominates. The fact that pulmonary valve closure is so well heard at the apex indicates considerable pulmonary hypertension. The diastolic rumble is intensified early in expiration.

(Below) apex in M. W. (618373) 33 years old and asymptomatic. The presystolic murmur is accentuated in inspiration and early expiration. OS interval is shortest during cycles with loudest diastolic murmur. All clinical and radiologic evidences point to mitral stenosis not tricuspid stenosis in this patient.

begin immediately with the opening snap but only after a brief interval. In some of such cases there is at the beginning of the murmur a concentration of vibrations which should be considered a sound. Since it has the temporal relationship of a third heart sound, I will so refer to it. It is difficult to

imagine there being a rapid filling sound arising in the left ventricle in MS in view of the impediment to ventricular filling. It may be a third sound produced in the right ventricle (1588, 1062). Contro (285) found a third heart sound in 5 of 84 cases of presumed mitral stenosis.

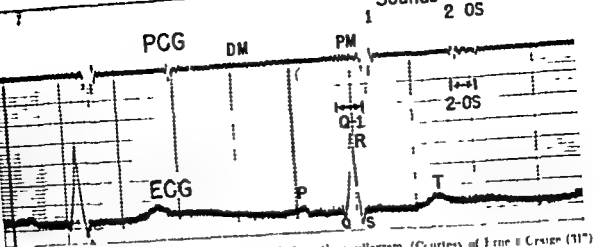


FIG 283 Measurement of the Q-1 and 2-OS interval from the phonocardiogram (Courtesy of Irene E. Craige (11)) and the New England Journal of Medicine)

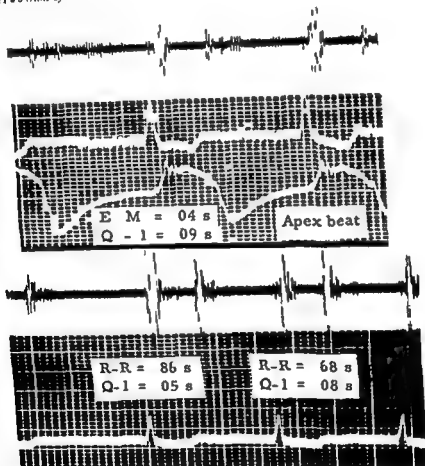


FIG 284 Measurement of Q-1 interval from the phonocardiogram (Courtesy of Kelly (11)) and Circulation (top) Period of time on a middle-aged man with proved mitral stenosis. The electromechanical interval (EM) demonstrated to be 0.04 sec by the systolic deflection of the apex impulse. This was confirmed by direct measurement of ventricular pressure at operation. In the absence of mitral stenosis the first sound occurs at this time. In this instance the first sound began 0.09 sec after the beginning of the QRS. Note the deflections of the apex beat simultaneous with the first sound (bottom) Trace obtained from subject with proved mitral stenosis. Atrial fibrillation with varying RP interval is present. When the preceding diastole is long the Q-1 is short and when diastole is short the Q-1 lengthens. A long diastole allows better left atrial emptying and consequently a lowering of the left atrial pressure.



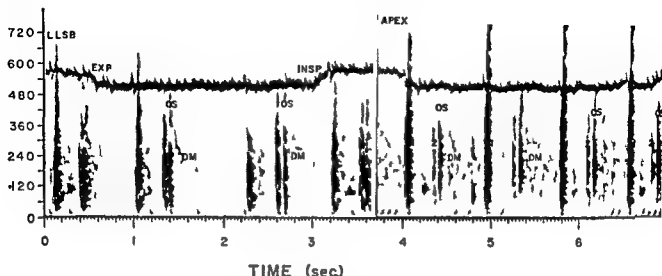


FIG. 282 Mitral stenosis with atrial fibrillation

LLSB (left) and apex (right) in P.M. (742071) 48 year old female. The S<sub>1</sub>-S<sub>2</sub> interval is longer after longer diastolic periods. S<sub>1</sub> is less loud after longer diastolic periods. The C features are not evident at the apex where the rate was more regular during the period of the recording. A peculiar musical murmur in a tone may be of pericardial origin. The diastolic murmur is unusually high pitched for mitral stenosis. Even the diastolic murmur at LLSB is probably of mitral origin.

is essentially an ejection stenosis murmur, and like that of aortic stenosis is diamond shaped and Christmasy tree shaped in the oscillogram and SPCG respectively. However when PR is of normal duration and atrial systole bears a normal relationship to ventricular systole, the Christmasy tree is as it were cut in half vertically and the result is the so called pre-systolic crescendo. When the PR interval is prolonged the full con-

the Gallavadin theories seem unlikely, however the billowing of the mitral curtain toward the atrium with ventricular contraction would be expected to be interrupted by snapping movement. Mitral regurgitation cannot be expected to precede the first sound because if there is a reversal of the polarity of atrioventricular pressure differential to produce regurgitation the reversal will produce the first heart sound. Either the Gallavadin or the Nichols theory would require that with atrial fibrillation one vibration would persist and be recorded between the onset of the QRS and S<sub>2</sub>. Such vibrations are not recorded at least the only vibrations which are recorded have an appearance consistent with an origin in closure of the tricuspid valve or in the contracting myocardium itself.

The lack of gap between the pre-systolic murmur and the first sound is perhaps not troublesome if one thinks of the pre-systolic murmur as one part of a bisected ejection stenosis murmur (i.e. half a Christmasy tree or diamond). The rising pitch which Brockbank (p. 20) felt was so difficult of explanation on the basis of an atrio-systolic murmur is likewise accounted for

figuration of the atrio-systolic murmur is revealed (Fig. 281). Creighton Bramwell (1933) pointed out that in such cases if the atrioventricular relationships are such that atrial systole falls in early diastole the atrio-systolic murmur is louder than otherwise. This is true probably because atrio-ventricular pressure gradient is greater at that time (pressure in the ventricle is lowest) atrial contraction is by Starling's law most forceful because the volume of blood contained in the atrium is greatest and the passive diastolic and atrio-systolic murmurs are summated. The pre-systolic murmur disappears with the advent of atrial fibrillation. At times with atrial fibrillation and a rapid ventricular rate there appears to be a pre-systolic crescendo but this is only an illusion created by the fact that the passive murmur continues right up to the snapping M<sub>1</sub>. Some beginning particularly with Lewis (1904) have in fact claimed that the crescendo character of the pre-systolic murmur is always an auditory illusion. They reached this conclusion by the fact that the oscillographic PCG often shows no crescendo of intensity possibly because of inadequate frequency response characteristics. The spectral PCG leaves no doubt that the crescendo is bona fide.

The two parts of the diastolic murmur are best



heard at the apex, often in a very confined area. However, occasionally in young subjects with small, thin chests and a loud diastolic murmur of mitral stenosis the murmur will be heard unusually widely, sometimes even in the aortic area, but in such instances the point of maximum audibility is still the cardiac apex.

The intensity of the murmur is dependent in part on the volume and, therefore on the velocity of flow across the stenotic orifice. When the patient is in congestive heart failure no murmur may be heard because of low flow. When severe pulmonary vascular disease develops the murmur may be considerably diminished. Or the association of pulmonary vascular disease on some other basis may obscure the diagnosis because of minimal or absent diastolic murmur. For instance in patients with severe chronic bronchial asthma and patients with severe pulmonary emphysema the presence of mitral stenosis may be long overlooked. The presence of emphysema in such cases impedes

transmission of any murmur which might be produced. Incidentally it is of further interest that the murmur may be heard only at an unusually low site, e.g., the left costal margin in the mid-clavicular line in such cases. Among a series of mitral stenosis with very high pulmonary vascular resistance reported by MacLennan, Wade and Vickers (1913), there was no diastolic murmur in three. Rivin (1248) described an interesting patient with myxedema and mitral stenosis in whom the latter diagnosis was not made until the myxedema was treated and mitral flow increased.

Jevins and Love (890) described 19 cases of mitral stenosis in which no murmur was present at one time or another. "True and totally mute mitral stenosis" must be very rare, rather it is usually the physician who is deaf. In the absence of a diastolic murmur the opening snap and the telltale change in the mitral first sound persist.

Complication is taken of the role of flow in the genesis of the murmurs of mitral stenosis by the several maneuvers (external and internal nitrate inhibition (888) etc.) employed to bring it out. All of which are designed to increase flow. Since the patient with severe mitral stenosis cannot increase his stroke volume—and his mitral flow—much of the effect of these measures is largely mediated through the tachycardia. The shortening of diastole creates the necessity for more blood to pass through the orifice in a given unit of time. With sinus rhythm there is to some extent a summation of the effects of passive flow with those of atrial systole.

The murmur of mitral stenosis is loudest in the recumbent position (especially in the left lateral decubitus which probably brings the apex more intimately in contact with the anterior chest wall) and for this reason favors transmission of the murmur. The act of turning into the left lateral decubitus many times is sufficient exertion to bring out the murmur in the first few of the cardiac cycles which follow. The usual practice is to have the patient exercise by sitting up and lying down several times in rapid succession. He then turns immediately into the left lateral decubitus suspending respiration if possible and the physician listens attentively for the telltale murmur. Because of its relatively low frequency

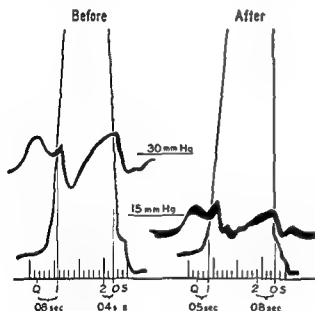


FIG. 285 Relation of left atrial pressure to Q 1 and 2 OS intervals.

Pressure recorded during operation from the left atrium and the ventricle in a patient with severe mitral stenosis. Q refers to the beginning of the QRS complex of the electrocardiogram. 1 to the first rapid vibration of the first sound. 2 to the second sound. OS to the opening snap of the mitral valve. Phonocardiograms were taken preoperatively and postoperatively when the heart rates were similar. (Courtesy of Kelly (779) and Circulation.)



Fig. 38. From top to bottom three curves in aorta, left ventricle, and left atrium recorded simultaneously with equisensitive manometers and on the same time line. (Left) Subject with normal circulation. (Right) Subject with mitral stenosis. (1) Onset ventricular contraction. (2) closure of mitral valve in MS. (3) opening of aortic valve. (4) isometric contraction. (5) maximum ejection. (6) reduced ejection. (7) aortic valve closure. (8) isometric relaxation. (9) opening of mitral valve. (10) rapid filling. (11) late systole. (12) atrial systole.

The large diastolic gradient in mitral stenosis is evident as is the reaction to the delayed MI and all resisted S<sub>2</sub>O<sub>2</sub> interval. In connection with general observations it is of interest that in the normal recordings atrial pressure falls below ventricular pressure at the point in the heart cycle when S<sub>2</sub> occurs ( courtesy of Braunwald et al. (1970) and C. C. Chou ).

conspicuous the murmur is best heard with the bell chest piece applied lightly to avoid the diaphragm effect of the underlying skin.

Careful search for a diastolic murmur is indicated when one hears a snapping, first heart sound and/or in opening snap both of which are rather frequently present in the absence of a diastolic murmur on routine auscultation. The presystolic portion of the diastolic murmur is the one most likely to be present when there is sinus rhythm and when it is a matter of only one being heard. Although pressure flow across the valve may not have sufficient velocity to generate a murmur the contraction of the atrium with active propulsion of blood through the obstruction especially because left atrial hypertrophy is usually present

produces sufficiently rapid mitral flow to result in a murmur.

Durozier (1921) invented the oronomatopoeic device for the sounds of the open mitral stenosis. The significance of each component of the device is indicated in Figure 274. The actual imitative usefulness of Durozier's device is destroyed if too unglorified a rendition is provided. Sound must be pronounced by expelling air at unusual high velocity past the tongue and through the lips and teeth. It is given a staccato or machine gun tempo. It has a rolling *rr*. Furthermore someone has referred to mitral stenosis as resulting in the "bowling alley heart" the second sound and opening snap correspond to the impact of the ball

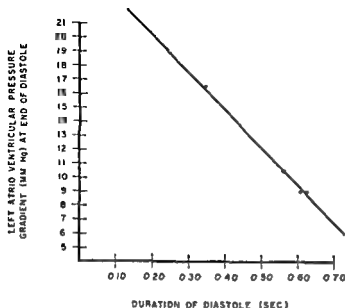


FIG 257 Relation between left atrioventricular gradient at end of diastole and duration of diastole in patient with mitral stenosis and atrial fibrillation. The greater the gradient the greater the Q1 interval can be expected to be. The gradient was measured from records similar to that in Figure 256B, however the patient had atrial fibrillation. (Courtesy of Braunwald et al. (169) and *Circulation*.)

on the floor and one bounce the rumble corresponds to the sound of the ball rolling down the alley, the snapping first sound is the impact of the ball on the pins.

An early systolic click generated in the dilated pulmonary artery is frequent in mitral stenosis. It is loudest in the pulmonary artery and may introduce a short murmur. This sound (p 129) may be produced by snapping of the arterial wall under the impact of ventricular ejection. The click usually occurs about 0.09 sec after the beginning of the QRS of the electrocardiogram. The click occurs with dilation of the pulmonary artery due to other causes. In the oscillogram and to a much lesser extent to the ear, the systolic click can be misinterpreted as a delayed snapping first heart sound. For example, in a patient with primary pulmonary hypertension or multiple emboli the radiologic findings may suggest mitral stenosis and the early systolic click may be taken for a snapping, delayed mitral first sound.

When pulmonary hypertension is severe a Graham Steell murmur of pulmonary regurgitation

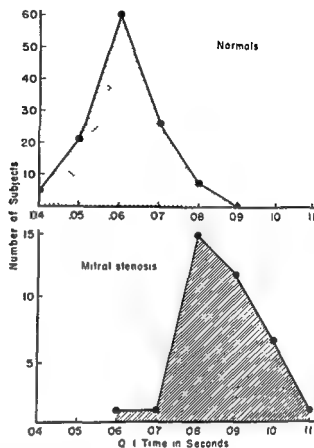


FIG 258 Q1 time in patients with mitral stenosis compared with that in normal subjects. (Courtesy of Craig (317) and the *New England Journal of Medicine*.)

may be present. It is heard at the left sternal border, is seldom loud, and at times (rarely in fact) shows accentuation with inspiration. Differentiation from the murmur of mild aortic regurgitation is usually difficult since peripheral signs of aortic regurgitation may be absent. Accentuation of the second sound in the aortic area suggests aortic valve disease (see p 172) and if the diastolic murmur is audible at all at the right of the sternum the murmur is probably of aortic origin (632).

After commissurotomy (281, 519, 1139) the diastolic murmur is diminished as a rule and occasionally is completely abolished. During the week or ten days immediately following operation the impression may be obtained that the murmur has completely disappeared. However when the patient becomes more active with increase in cardiac output and therefore in mitral flow the murmur is found to be still present. The first sound

remains snapping (142) unless a great deal of mitral regurgitation has been produced but the Q1 delay is reduced likewise the opening snap usually persists but the S<sub>2</sub>-OS interval is increased P<sub>2</sub> may be diminished in intensity and a Graham Steell murmur may disappear Occasionally an associated valve lesion which was not suspected before operation or was deemed insignificant blows forth after relief of the mitral obstruction Specifically the aortic stenosis may become clearer (Fig. 300)—the patient may tire poorly after mitral valvulotomy if the aortic obstruction is not also relieved—and those of tricuspid stenosis (Fig. 320) may also to some extent be accentuated After operation there may be both an atrial gallop and presystolic murmur recorded at the apex Atrial gallop alone have occurred frequently The atrial

sound sometimes has the characteristics of a snap (see Figure 297) Production of such a presystolic snap through collaboration of the left atrial hypertrophy and residual fibrosis of the mitral mechanism is perhaps not surprising The sound may disappear after operation Atrial pleuropericardial adhesions cannot be excluded as a mechanism for the sound

Phonocardiograms before and after mitral valve surgery are not only within the abilities of every institution in which valve surgery is done but are in my opinion absolutely mandatory in the minimum pre- and post-operative evaluation Repeated phonocardiographic observation with attention to the indices of severity of mitral stenosis constitute one of the best methods of postoperative detection of stenosis (1072) (Fig. 274)

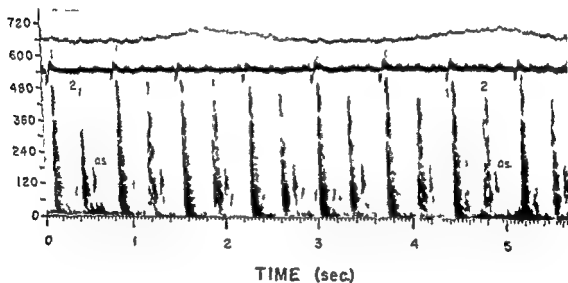


FIG. 280 The influence of atrioventricular pressure gradient on S<sub>2</sub>-OS interval. The upper recording is from the pulmonary area of a patient with severe aortic hypertension and a mild degree of mitral stenosis (Upper line = respiration mark with upward motion indicating inspiration downward motion indicating expiration). The second sound becomes split with inspiration. In spite of the systemic hypertension the pulmonary artery pressures are predominantly diastolic. Of particular note is the unusually great S<sub>2</sub>-OS interval measured 0.14 sec from beginning of S<sub>2</sub> to beginning of opening snap in two cycles. Other clinical evidence indicates only a low grade of mitral obstruction in this case. A second factor in the S<sub>2</sub>-OS prolongation in this particular case is the systemic hypertension. The patient's blood pressure were 220/140 mm Hg at the time of this recording suggest that aortic valve closure might occur perhaps at 150 mm Hg and that an appreciably longer time will be required for intraventricular pressure to fall to the level of intra-atrial pressure at which time the opening snap will occur. The considerations are graphically presented in the graph on the next page (Fig. 280 B).

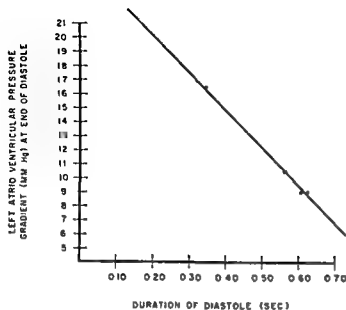


FIG 267 Relation between left atrioventricular gradient at end of diastole and duration of diastole in patient with mitral stenosis and atrial fibrillation. The greater the gradient, the greater the Q1 interval can be expected to be. The gradient was measured from records similar to that in Figure 266B, however the patient had atrial fibrillation (Courtesy of Braunwald *et al* (169) and *Circulation*).

on the floor and one bounce, the rumble corresponds to the sound of the ball rolling down the alley; the snapping first sound is the impact of the ball on the pins.

An early systolic click generated in the dilated pulmonary artery is frequent in mitral stenosis. It is loudest in the pulmonary area and may introduce a short murmur. This sound (p. 129) may be produced by snapping of the arterial wall under the impact of ventricular ejection. The click usually occurs about 0.09 sec after the beginning of the QRS of the electrocardiogram. The click occurs with dilation of the pulmonary artery due to other causes. In the oscillogram and to a much lesser extent to the ear, the systolic click can be misinterpreted as a delayed snapping first heart sound. For example, in a patient with primary pulmonary hypertension or multiple emboli, the radiologic findings may suggest mitral stenosis and the early systolic click may be taken for a snapping, delayed mitral first sound.

When pulmonary hypertension is severe, a Graham Steell murmur of pulmonary regurgitation

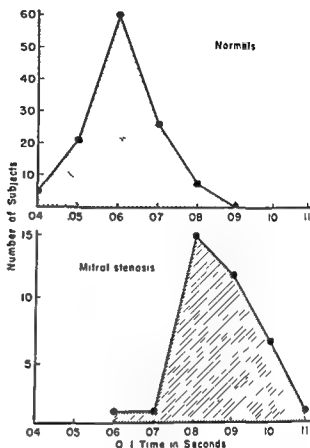


FIG 268 Q1 time in patients with mitral stenosis compared with that in normal subjects (Courtesy of Crage (317) and the *New England Journal of Medicine*).

may be present. It is heard at the left sternal border, is seldom loud, and at times (rarely, in fact) shows accentuation with inspiration. Differentiation from the murmur of mild aortic regurgitation is usually difficult, since peripheral signs of aortic regurgitation may be absent. Accentuation of the second sound in the aortic area suggests aortic valve disease (p. 172) and if the diastolic murmur is audible at all at the right of the sternum, the murmur is probably of aortic origin (602).

After commissurotomy (251, 319, 1139) the diastolic murmur is diminished as a rule and occasionally is completely abolished. During the week or ten days immediately following operation the impression may be obtained that the murmur has completely disappeared. However, when the patient becomes more active with increase in cardiac output and therefore in mitral flow, the murmur is found to be still present. The first sound

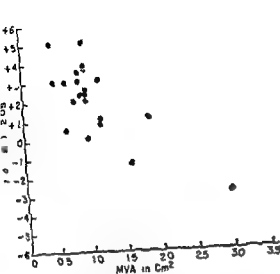


FIG. 26 Relation of Wells index ( $Q_1$  minus  $2 Q_2$ ) to the mitral valve area (MVA) as determined at operation or at cardiac catheterization by Gorlin's formula (Courtesy of Craige [31]) and the *New England Journal of Medicine*.)

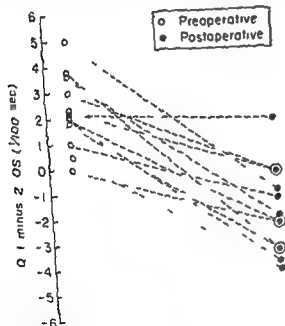


FIG. 27 Wells index in 10 patients before and after valvulotomy for mitral stenosis (Courtesy of Craige [31] and the *New England Journal of Medicine*.)

the diastolic murmur is the generally accepted near pathognomonic indication of mitral stenosis; the most worthy by far for such a hint.

Mitral regurgitation is included in the list, in view of a certain minor degree of mitral closure

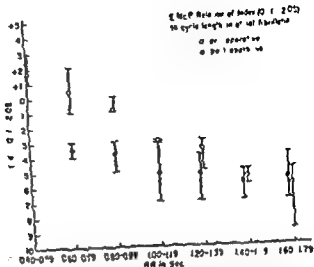


FIG. 28 Relation of Wells index to cycle length in a patient with mitral stenosis and atrial fibrillation before and after mitral valve surgery.

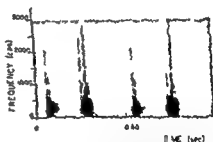
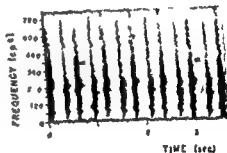


FIG. 29 (above) Pulmonary area in patient with high grade mitral stenosis. There is a faint (rather weak) murmur (GS) (below) same area. The recording traces how it is possible to spread out the time base but with the original spectrographic method only at the expense of frequency axis. In this instance the heart sound is sufficiently intense that, especially with additional amplification two components are seen in the first sound and three in the second. It is uncertain whether the components of the first sound are tricuspid and mitral closure sound or only one of the sounds plus an early ejection snap. There is a short early systolic murmur. The three components in the region of  $S_2$  are thought to be aortic closure sound, pulmonary closure sound, and mitral opening snap in this order.



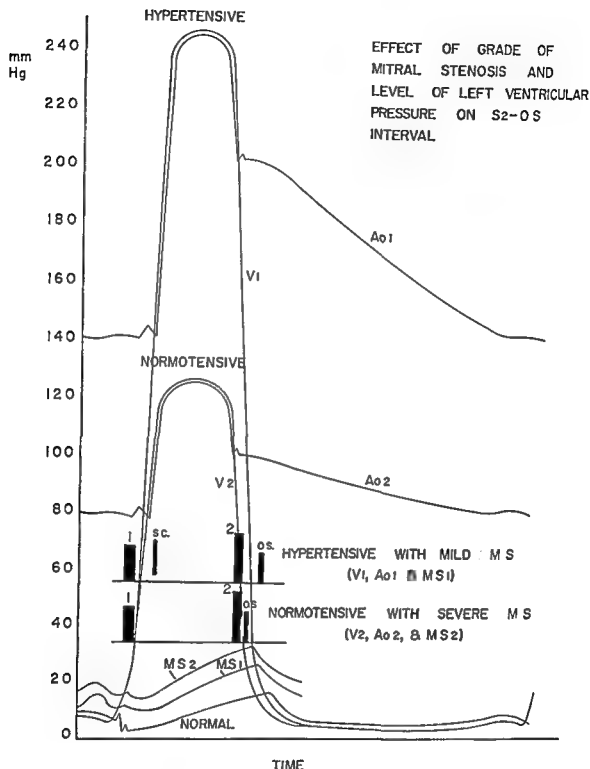


Fig 290 B

### The Auscultatory Simulation of Mitral Stenosis

All that rumbles is not mitral stenosis  
Hurst (734)

The list in Table 12 is a partial enumeration of the conditions which produce auscultatory signs

similar to the diastolic murmur of mitral stenosis. The list could be extended by mention of conditions in which a snapping M<sub>1</sub> may be heard (p 286) in which pulmonary early systolic click and accentuated P occur (p 289) in which the S<sub>2</sub>-O<sub>2</sub> combination is imitated (p 290) and so on. But

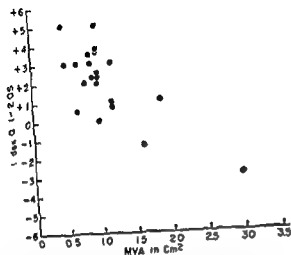


FIG 20 Relation of Wells index ( $Q_1$  minus  $2 OS$ ) to the mitral valve area (MVA) as determined at operation or at cardiac catheterization by Gorlin's formula (Circulation of Craige (31) and the New England Journal of Medicine)

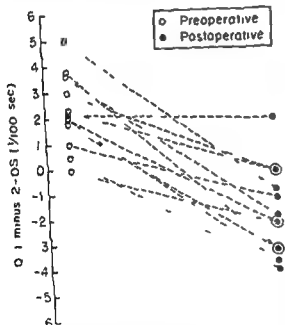


FIG 21 Wells index in 12 patient before and after valvulotomy for mitral stenosis (Circulation of Craige (31) and the New England Journal of Medicine)

the diastolic murmur as the generally accepted near pathognomonic indication of mitral stenosis is the most worthy basis for such a list (Mitral regurgitation) included in the list since given a certain minor degree of mitral obstruc-

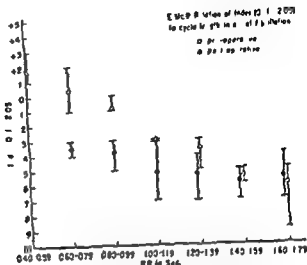


FIG 22 Relation of Wells index to cycle length in a patient with mitral stenosis and atrial fibrillation before and after mitral valve surgery

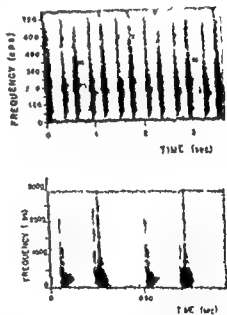


FIG 23 (Upper) Pulmonary area in patient with high grade mitral stenosis. There is a faint Graham Steell murmur (GS) (Heter) same area. The record illustrates that it is possible to spread out the time element with the original spectrographic method only at the expense of frequency gain. In this instance the heart sounds are sufficiently intense that especially with a fractional amplification two components are seen in the first sound and three in the second. It is uncertain whether the components of the first sound are triphasic and if mitral closure sounds or only one of the components plus an early aortic snap. There is a short early systolic murmur. The three components in the region of  $S_2$  are thought to be aortic closure and pulmonary closure sound and mitral opening snap in this order.

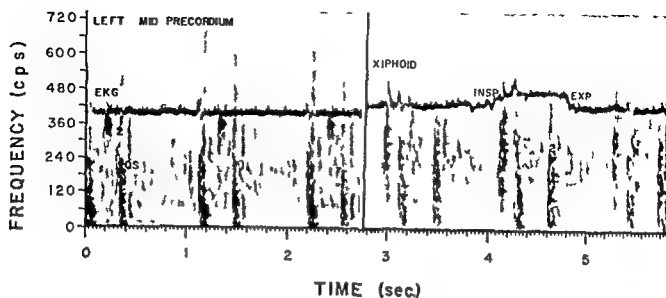


FIG 204

FIG 205

Figs 204 and 205 Systolic squeak of pericardial origin and atrial pericardial friction early after mitral valvulotomy

II C (30556) 35 years old demonstrates (in the e recordings made one week after mitral valvulotomy) a late systolic squeak over the left midprecordium and a presystolic (atrial) friction sound over the xiphoid. The  $S_2$  OS interval is prolonged

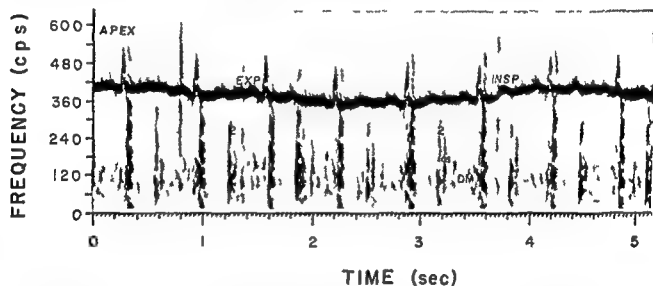


FIG 206 Effects of commissurotomy

The typical findings of mitral stenosis present before operation in I C (754164) are shown here. After operation (the recording is not shown)  $S_1$  was less delayed and the  $S_2$  OS interval was increased. An atrial friction rub was now present.

tion the presence of mitral regurgitation will exaggerate the murmur which would be heard if no regurgitation were present. The reason is that regurgitation produces increased mitral flow. Not only must something approaching a normal quantity of blood pass the valve but also that volume of blood regurgitated during the previous ventricular systole. It is possible to imagine a degree of mitral

stenosis which would produce no murmur were it not for the coincidence of mitral regurgitation.

The Carey Coombs murmur of early rheumatic mitral valvulitis does not indicate permanent or reversible valve damage. It is usually introduced by a third heart sound and is a blubbery mid diastolic murmur which usually has no presystolic accentuation despite the presence of sinus

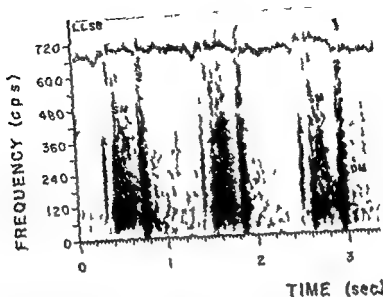


FIG. 10. ILBBINMITR (35RM) six months after surgical correction of aortic stenosis. Note (1) relatively dull  $S_2$  (decreased snapping before operation) (2) decreased aortic murmur (3) a broad second sound which probably is due to aortic regurgitation (4) not at all present is a third heart sound initiating a short diastolic murmur (all in locations of mitral regurgitation) (5) probably is created at surgery. Clinically the patient has improved. Note (1) huge E wave of ECG (polarity inverted) and (2) the striking presystolic gallop which looks and sounds like a presystolic snap. The latter is a fairly frequent postoperative finding.

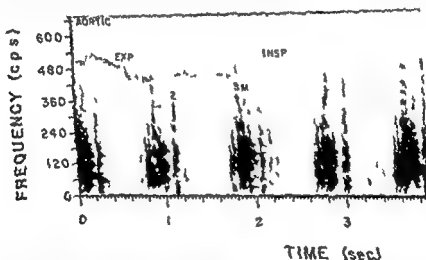


FIG. 11. Regurgitation of murmur of A after surgical correction of AS.

In 1960 (180606) the aortic murmur of the aortic area was not improved before operation but provided unmistakable evidence of AS after operation as shown here.

rhythm. The reversible nature of the murmur is consistent with the view that it is due to edema in the valve plus dilatation of the ventricle. The latter is perhaps not central since the murmur may occur in a heart which does not seem enlarged. Linn and Durr (1948) point out with

apparent validity that diastolic filling of the ventricle is more likely to be accompanied by sound in children with aortic phylogenic  $S_2$ . From the standpoint of an eubiotic characteristic the Carey Coombs murmur is the prototype of the murmur of relative mitral stenosis of

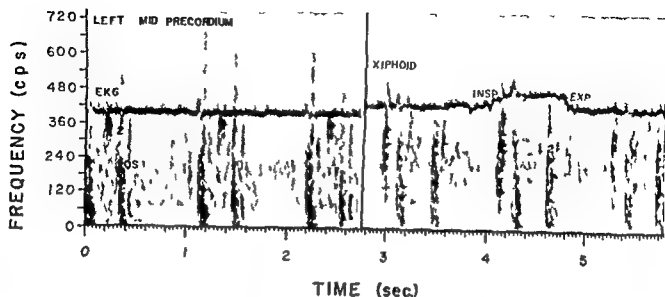


FIG 204

FIG 205

Figs 204 and 205. Systolic squeak of pericardial origin and atrial pericardial friction early after mitral valvulotomy

B C (305550) 35 years old demonstrates (in the e recordings made one week after mitral valvulotomy) a late systolic squeak over the left midprecordium and a pre-systolic (atrial) friction sound over the xiphoid. The S<sub>1</sub>-S<sub>2</sub> interval is prolonged

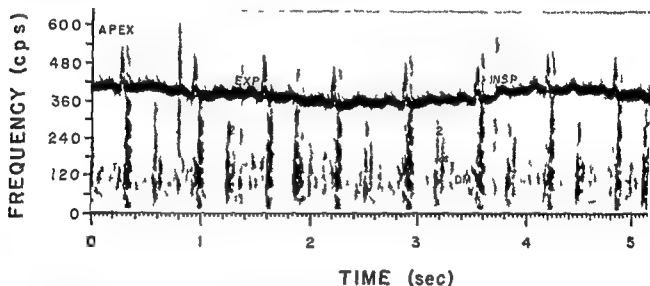


FIG 206 Effects of commissurotomy

The typical findings of mitral stenosis are present before operation in B C (754164) are shown here. After operation (the recording is not shown) S<sub>1</sub> was less delayed and the S<sub>2</sub>-S<sub>3</sub> interval was increased. An atrial friction rub was not present.

tion the presence of mitral regurgitation will exaggerate the murmur which would be heard if no regurgitation were present. The reason is that regurgitation produces increased mitral flow. Not only must something approaching a normal quantity of blood pass the valve, but also that volume of blood regurgitated during the previous ventricular systole. It is possible to imagine a degree of mitral

stenosis which would produce no murmur were it not for the coincidence of mitral regurgitation.

The Carey-Coombs murmur of early rheumatic mitral valvulitis does not indicate permanent or reversible valve damage. It is usually introduced by a third heart sound and is a blubbery mid-diastolic murmur which usually has no pre-systolic accentuation despite the presence of sinus

murmur. In 2:1 W diastolic position possibly in elderly persons in particular a Carey-Coombs murmur may occur in early diastole if the heart rate is proper. A combination of rapid ventricular inflow and atrial systole may be possible. With the second P wave (which was followed by a ventricular contraction) there may be no murmur.

Anemia, particularly chronic anemia such as that of hook worm disease and of sickle cell disease is a notorious mimic of mitral stenosis. (See the more detailed discussion on pp. 447 to 449.)

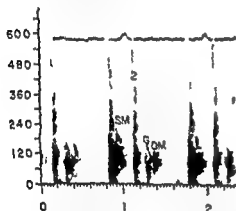


FIG. 99. Carey-Coombs murmur at apex after exercise in 14 year old patient with acute rheumatic fever (L. F. A. 492). During a course of cortisone in high dosage the gallop and rumble became fixed even at rest.

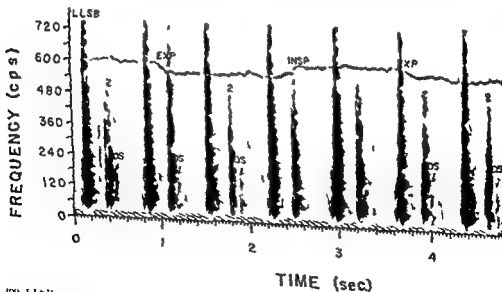


FIG. 100. LLSB; recorded in L. B. 1411/223, a 14 year old female with myxoma of left atrium which was surgically removed. Single tracing of a pre-systolic murmur and of a presystolic snap are seen. Both the first and second sounds are accentuated and the murmur is moderately fixed in relation to the QRS.

$M_1$  is likely to be snapping and there may be a presystolic murmur or even a diastolic murmur beginning in the earlier part of diastole. Dilatation of the ventricle, accelerated velocity of flow, increased cardiac output and reduced viscosity of blood are the factors operating in the complex causation of these phenomena.

Figure 301 presents the crucial autopsy finding in a patient thought clinically to have mitral stenosis and regurgitation on a rheumatic basis. Autopsy revealed suppurative endocarditis with a major complication of the posterior aortic cusp such that regurgitation of blood against the aortic leaflet of the mitral valve was likely to have occurred. Displacement of the aortic leaflet by the regurgitant stream from the aorta has been a favorite possibility for the mechanism of the Austin Flint murmur. Rarely does one get such convincing evidence as in this case. (Oulley (81) and Lalwani and Burchell (112) presented an autopsy specimen in which a jet lesion developed on the aortic leaflet of the mitral valve clearly due to the stream of blood regurgitating against it through the aortic valve (see page 283 for further discussion of the Flint murmur).)

In constrictive pericarditis I have observed a Carey-Coombs type of murmur in two patients after operation (Fig. 432). I have no clear idea of

TABLE 12

*Conditions with auscultatory signs simulating the diastolic murmur of mitral stenosis\**

- I Conditions producing diastolic rumble at the apex
  - A Mitral regurgitation (see text)
  - B Carey Coombs murmur of early rheumatic mitral valvulitis
  - C High mitral flow (with dilated left ventricle)
    - 1 In congenital heart disease with left to right shunt
      - a Ventricular septal defect
      - b Patent ductus arteriosus
      - c Pulmonary arteriovenous fistula
    - 2 In complete heart block
    - 3 In anemia (1108)
    - 4 ? In thyrotoxicosis
- D Tricuspid stenosis
  - 1 Rheumatic
  - 2 Relative
    - a In atrial septal defect or anomalous pulmonary venous return
    - b With pulmonary hypertension as in mediastinal collagenosis (J B 66434) multiple pulmonary emboli (D D 745702) etc (336 1263 1279 1595)
- E Austin Flint murmur accompanying aortic regurgitation (p 283)
- F Constrictive pericarditis
- G Myxoma of the left atrium
- H Malformation of left ventricular myocardium (336)
  - I More dilatation of the left ventricle as in congestive heart failure of any cause (1287 1520) probably especially if flow is relatively well maintained, as in so called high output failure
  - J Coarctation of the aorta (p 306)
  - K Congenital aortic stenosis (p 399)
- II Conditions mistaken for diastolic murmur
  - A Icteric gallop
  - B Eccentric construction of first heart sound (107 738)
  - C Protodiastolic gallop

\* See references 154 345 986 992 1520

most of the examples in I of table 12. I or example at times the term Carey Coombs is applied to the mitral murmur of ventricular septal defect (1590). The occurrence of this murmur which is sometimes very striking in association with congenital malformations (and the murmur of relative tricuspid stenosis with atrial septal defect) often raises the question as to whether congenital mitral stenosis is also present. In atrial septal defect<sup>6</sup>

\* See p 349 for a discussion of the numerous other respects in which ASD may simulate MS by auscultation

TABLE 13

*Causes of mitral regurgitation*

- A Affecting the cusps predominantly
  - I Anatomic
    - 1 Rheumatic
    - 2 Bacterial endocarditis
    - 3 Congenital
    - 4 Traumatic
  - II Functional
    - 1 Atrial fibrillation
    - 2 Myxoma or ball valve thrombus (p 210 and 220)
- B Predominantly affecting the valve seat and suspension (i.e. the chordae and/or fibrous and muscular annulus)
  - I Ruptured papillary muscle or chordae tendineae
  - II Calcified annulus fibrosus mitralis
  - III Dilated ventricle on the basis of myocardial disease or the strain of systemic arterial hypertension or disease of the aortic valve

because of dilatation the right ventricle constitutes the apex, and the murmur of relative tricuspid stenosis is heard there. Even rheumatic stenosis of the tricuspid valve may be accompanied by a murmur heard fairly well out toward the apex. In one case (I T 315167) of large pulmonary arteriovenous fistula (1303) there was a Carey Coombs murmur in the mitral area. In location and quality it was quite distinct from the typical continuous murmur of the fistula itself.

In complete heart block, Wood (1590) states that a diastolic murmur may be heard. In over three fourths of the patients with congenital heart block of complete degree a functional mitral diastolic due to the large mitral stroke blood flow was heard, particularly when the rate was under 80. Among 22 cases of congenital complete heart block referred to by Nadas (1137) half had an early or mid diastolic rumble. Rydand (1336) described an atriodiastolic murmur in complete heart block in elderly persons. The ventricle is necessarily dilated in complete heart block to accommodate the increased stroke volume. Furthermore although the flow is spread over a longer period of time diastolic mitral flow is increased in volume and probably is particularly rapid during the ventricular filling phase of early diastole. I or these two reasons the occurrence of a functional diastolic murmur would not be surprising. See p 442 for a discussion of the mechanism of Rydand's atriodiastolic

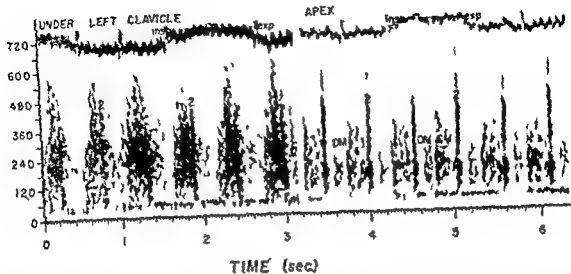


FIG. 303 Recorded under left clavicle and at apex in B. A. (1931) with patent *f. clus. anterior* is proximal to coarctation. In the former there is a typical continuous murmur with peak in late systole. In the latter there is a mid-diastolic rumble due probably to "torrential" mitral flow. The combination of coarctation with *f. clus. anterior* exaggerates the left to right heart. A coarctated fibrous to it with mitral valve involvement is possible but it is necessary to account for the murmur at the apex (aortic closure murmur) is ringing. Hypertension may be the cause of the coarctation but a coarctated aortic valve disease (frequent with coarctation) is to be suspected.

present when the patient is upright and is tolir when supine. The first finding rather pathologic of left atrial tumor or fibrous valve thrombosis was described in 1894 by Wilson (1193) and in 1917 by Mörz (101). In his monumental work on tumor of the heart Mahoney (1023) referred to the phenomenon as "paradoxical mitral stenosis" since the murmur of ordinary mitral stenosis tends to disappear when the patient is upright.

Davis and Andrus (336) described one patient with what they termed mitral stenosis in the infant produced by a congenital malformation of the myocardium of the left ventricle and a second patient with median mitral collagenosis. In both M<sub>1</sub> and I were increased and there was a mid-diastolic rumble at the apex. In the second the diastolic murmur may have been of tricuspid origin (see p. 328). One case described by Teare (1414) under the title "A symmetrical hypertrophy of the heart in young adult" seems to have been the same condition as in the first case of Davis and Andrus. Teare's case was also subjected to operation for presumed mitral stenosis.

Occasionally a diastolic rumble is present during heart failure and disappears with restoration

of compensation (161). I observed this sequence of events in a 76-year-old man with congestive failure and atrial fibrillation on the basis of coronary arteriosclerosis. I recall the sequence of events in true mitral stenosis is the converse: the diastolic murmur is likely to become inaudible during failure. Merely dilation of the ventricle without failure necessarily may result in a diastolic rumble suggesting mitral stenosis as in a 27-year-old individual with coronary artery disease who was subjected to mitral valvulotomy (see Fig. 453 p. 322). Although obviously not unique to Endreth's case, one can point to the mid-diastolic and presystolic murmur which has been described (312, 662, 1212, 1339, 1185) in association with the myocardiopathy of this primarily neurologic disorder as another example of the mutation of mitral stenosis by myocardial disease. Atrial dysrhythmias also occur fairly frequently in these cases, increasing the mutation of rheumatic heart disease in these young individuals in whom the involuntary movements may be thought to represent "systemic chorea" and nothing of the legs occur commonly.

In the early part of the first heart sound there is often a low pitched component formerly



the mechanism, although dilatation of one or both ventricles was probably present in both and the first approximation to an explanation would appear to be dilatation of the ventricle, resulting from partial disuse atrophy and some fibrosis, in association with AV rings which are normal in dimension and possibly are kept normal by scar tissue in the AV groove. Also without operation in constrictive pericarditis a mid diastolic murmur has been described (263, 461).

With one possible exception, myxoma of the left atrium (and ball valve thrombus (1003),

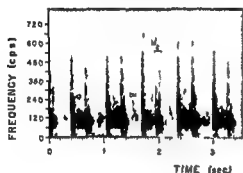


FIG 301 Mid diastolic rumble at apex with atrial septal defect (M M A76411). Split S<sub>2</sub> also characteristic of ASD

which usually occurs only when there is mitral valve disease with an element of stenosis) can produce all the auscultatory signs of mitral stenosis—delayed, snapping, M<sub>1</sub>, diastolic rumble at the apex, accentuated P<sub>2</sub>. The possible exception is the mitral opening snap (1023). However, a diminutive sound of this type may be demonstrable (see Fig 302). In a case of myxoma with phonocardiograms of presumed opening snap recently published (868), the interval between S<sub>1</sub> and the extra sound is consistent with the interpretation offered. However, the phonocardiogram demonstrates a longer sound than the opening snap usually is. Possibly it introduced a short rumble. In another report (261) it is stated that "an opening snap was evident" stethoscopically. The mitral valve was normal at autopsy. In his patient Ludwig, (970) described the presence of *Wachtelschlag* sound of the pul. Since this was a simile used by German writers (1214) for the second sound and opening snap following in close succession it has been assumed by subsequent authors (1023) that an opening snap was present in that case. As to the murmur of myxoma some (116, 824, 1023) have claimed it is diastolic and

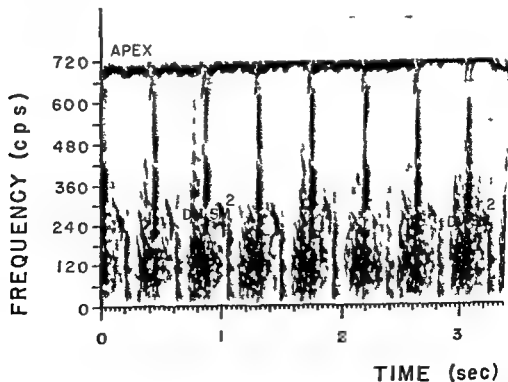


FIG 302 Apex in D T (B6445) with *B communis*. Note diastolic murmur which because of the rapid rate is a summation of passive and active murmurs. Evidences of light murmurality of the systolic murmur (see Fig 187)



Fig. 30. Traumatic mitral regurgitation. Autopsy case (6) of a 29-year-old man who died in Winkels' clinic in Vienna in 1924 ten years after attempting suicide with a revolver. The bullet entered in the fourth intercostal space two fingerbreadths below the nipple. He was seen in the Surgical Clinic and operated on. In the left pneumothorax and a small hemorrhagic effusion were found. The full thickness of the mitral valve was not recovered.

Rheumatic damage affects most commonly the mitral valve (see p. 261) and mitral regurgitation is the most frequently occurring sequel of rheumatic fever. It is this type of mitral lesion furthermore which persists only for millions of its high frequency is most often affected in subacute bacterial endocarditis. Pure mitral regurgitation unlike pure mitral stenosis is more frequent in men.

Congenital mitral insufficiency occasionally is found alone but usually is associated with another malformation, most often the ostium primum variety of atrial septal defect.

Traumatic mitral regurgitation is indeed rare. An instructive and remarkable case is that of a Viennese man (6) who in 1914 attempted suicide with a pistol and survived until 1924 dying of the effects of pure mitral regurgitation from a bullet hole in the anterior (anterior) leaflet of the mitral valve (see Fig. 30).

It is because the valve leaflets and chordae tendineae are so important to the competence of the AV valves that mitral regurgitation may occur with a great variety of conditions which in some way or ways affect the noncupular portion of the valve mechanism.

Following myocardial infarction a papillary muscle may rupture with resulting mitral regurgitation. Myocardial infarction and tearing of the papillary muscle may also result in mitral regurgitation through failure of the mechanism by which the

thereafter the patient showed a grating, apical systolic murmur and a loud systolic murmur in the pulmonary artery. Relative tricuspid insufficiency, atrial fibrillation and congestive failure developed.

The section shown here was made after allowing the specimen to fix for 48 hr. in formalin (above). The anterior portion of the first of the oval holes in the anterior cup of the mitral valve mechanism is shown. Both atria (the septum and part of the right atrium with entering venous cavities are seen) were tremulous by dilated and compressing the right lung. In fact the heart extended practically from right axilla to left axilla. The dilated hepatic veins and an enlarged liver. Chronic passive congestion are seen.

(Below) Anterior portion of the section cut. The left ventricle is seen in this view as well as in the prior one. The interatrial septum and the aorta are visualized. The bullet was found embedded in scar tissue in the region of the apex of the left ventricle. Again the tremendous atrial dilatation is evident.

thought to be of atrial origin (1166, 1241)—and indeed they may be in many cases, as discussed on p. 126—but considered to arise sometimes from contraction of the ventricular myocardium, since it was demonstrated (308) that it may persist in atrial fibrillation. When particularly striking, the low pitched “initial vibrations” followed by a sharp valve closure sound can suggest a presystolic murmur and accentuated  $M_1$  of mitral stenosis (536). Brimwell and Ellis (157) made the interesting auscultatory observation, which unfortunately did not have graphic documentation and analysis, that in 12 of 192 athletes there was a “curious prolongation of the first heart sound, not unlike the crescendo murmur of mitral stenosis.” Three were Marathon runners, three were long distance runners and three were

cyclists. The phenomenon was not present in any of 18 sprinters or 16 middle distance runners. In 1909 Sewall (1378) described “first sounds beginning with a crescendo tone, simulating closely the faint and brief presystolic murmur.”

#### MITRAL REGURGITATION

(Syn. Mitral insufficiency, mitral incompetence, MR)

#### ETIOLOGIC AND ANATOMIC CONSIDERATIONS

(191) Although the etiologic possibilities for true mitral stenosis are really only two (with many simulating conditions), the causes of mitral regurgitation are numerous (see Table 13, p. 196). Relatively the same situation exists in connection with aortic stenosis and aortic regurgitation (see p. 269).



FIG. 301 S. A. S. (269149) A 41-year-old female had had congestive heart failure for two months and murmurs interpreted as those of mitral stenosis and regurgitation, presumably rheumatic. At autopsy there were no stigmas of rheumatic fever but the aorta showed florid changes of syphilitic aortitis. There was a “sugar-scoop” deformity of the posterior cusp of the aortic valve, a positioned as to direct blood against the aortic leaflet of the mitral valve which shows a jet lesion just below the prolapsed cusp of the aortic valve.



FIG. 30° RAO in MR

P. M. (5/9/50) age 77 years had several bouts of acute rheumatic fever. There was grade IV systolic murmur; a diastolic murmur was heard only occasionally. An I questionably I V film (A) shows a mitral configuration of the heart with calcification in the left atrial appendage. In the lateral film the entire posterior wall of the left atrium is calcified and RAO in the lateral position (B) shows an extensive backward movement of the atrial wall with ventricular systole. In the enlarged view (C) left atrium (A) is above and left ventricle (V) below. In the area of the atrium an indentation produced by atrial systole is seen before the large outward (i.e. backward) movement with ventricular systole. Calcification provides the heart chambers with their own opaque markers for kymographic studies; conversely RAO is a good technique for demonstrating calcification because movement of the heart is less likely to blur the image—the exposure time in a given portion of a given frame is brief.

A

V

C

hot gun or more specific fixation for febrile illness. Healed bacterial endocarditis is likely to become a more frequent primary cause of regurgitation at the heart valves or important exaggerating factor in deformities already present.

Hepper and colleagues (674) describe a 79-year-old woman in whom a high-systolic murmur could be accounted for on the basis of healed and calcified bacterial lesions of the mitral valve.

In autopsies on 177 individuals 40 years of age



FIG. 306. Calcified annulus fibrosus mitralis in 88-year-old woman (M. S. 481084) with rheumatic fever in childhood (A). Exposure time 1.60 sec (B and C) RKA. In the enlarged view (C) it is seen that the calcified zone and the ventricular border move toward each other in ventricular systole. The movement of the base of the heart, as labelled by the calcified mitral ring, is of large amplitude. Descent of the base of the heart in ventricular systole was demonstrated by Leonardo da Vinci (774). Percutaneously he introduced three needles in the heart of a living animal, one at the apex, one at the base, one in the middle. With each ventricular systole the middle needle moved little, whereas the external ends of the apical and basilar needles moved away from each other. Systolic descent of the base has been demonstrated by others (1453) in patients with calcified mitral annulus and in animals in which radiopaque markers were placed in the heart (1531). An unusually loud, harsh, holosystolic murmur was present in this patient. A protodiastolic gallop characteristic of mitral regurgitation was also demonstrated.

papillary muscle shortens the effective length of its attached chordae during ventricular systole.

Mitral regurgitation occurs with calcification of the annulus fibrosus mitralis (Fig. 306), a condition which is poorly understood from the standpoint of pathogenesis, but which occurs in older people (1337) and sometimes in conditions favoring metastatic calcification, such as multiple myeloma. Rheumatism sometimes seems to be

involved in its causation. Complete heart block is sometimes associated. The rigidity of the fibrous skeleton apparently interferes with contraction of the muscular ring which ordinarily serves a significant role in narrowing the AV orifice and assisting competent AV valve closure. It is interesting that the tricuspid ring is so rarely involved relative to the mitral ring.

With the widespread use of antibiotics in

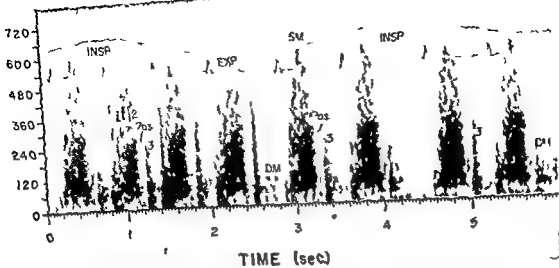


FIG 310 Rheumatic mitral regurgitation

In this case the systolic murmur begins with the second component of the first sound (mitral closure). The first sound is dull. In this case the systolic murmur although holosystolic is decelerating rather than a decelerating. A striking third heart sound gallop is present. This shows variation with respiratory phase being more conspicuous during expiration. It is followed by a short low pitched murmur (rattle). In the exp between the second and third sound there is a faint sound. That this is an opening snap and not the second part of a split second sound is suggested by the fact that its relationship to the second sound shows no variation with respiration. The presence of an opening snap in record as first as this for demonstrating it is not necessarily evidence that significant mitral stenosis is present. On the other hand its absence would be strong evidence against the existence of significant mitral stenosis.

motion of aortic valve Douglas (367) points out that aortic regurgitation of predominant mitral regurgitation may result from scarring and shortening of the chordae tendineae which hold the cusps open during ventricular systole. In such case the cuspal curtain may be mobile and the paradoxical accentuated.

The systolic murmur of mitral regurgitation is thought to be holosystolic in the great majority of cases. The rise and fall of interventricular pressure are rapid with most of systole spent at or near peak pressure. Reason to expect a holosystolic murmur with mitral regurgitation is suggested by Laster (213) thought dilatation of the heart might result in a non holosystolic early or late systolic murmur. This is questionable however as pointed out on page 290 an early decelerating non holosystolic murmur occurs rather commonly in cases of mitral stenosis. Although the mechanism is not entirely clear the murmur probably is produced by mitral regurgitation it may be that left ventricular pressure early in systole is sufficiently higher than the

pressure in the recently although only partially decompressed left atrium to produce mitral regurgitation but that as left atrial inflow continues and after the effects of descent of the base of the atrium are cancelled out there is inadequate pressure gradient to produce continued regurgitation and murmur.

The holosystolic murmur may be plateau decelerating or decelerating. The anatomic hemodynamic basis for these three different patterns is not clear. Particularly it is hard to understand the basis for a murmur which has its greatest intensity late in systole (plateau murmur) explicable thus is the usual pattern with high grade mitral regurgitation. A decelerating pattern probably results from the fact that although pressure in the left ventricle is relatively fixed during ventricular systole pressure in the atrium increases during the course of systole.

The systolic murmur is usually loudest at the cardiac apex and is well transmitted to the left axilla. With mammoth left atrium especially in young patients with small chests it is well heard

Movitt and Gerstl (1126A) questioned the benignity of pure mitral regurgitation when they reported autopsy studies of four men who died of heart failure from this lesion at the age of 42-50, 51 and 56 years. Olsen and Warburg (1153) followed up 14 persons under 30 years of age in whom a loud apical systolic murmur was the only finding. The average period of follow up was 11.8 years. Three of the patients had died.

Since the development of mitral valvulotomy for mitral stenosis, mitral regurgitation has taken on a more serious aspect. The association of mitral regurgitation in significant proportions usually has the effect that the benefit of surgery for the mitral stenosis is less than maximal. In addition, it is now appreciated that there is a small minority of cases of pure mitral regurgitation of such severity that it is a major source of embarrassment to the circulation (see above).

In mitral regurgitation in contradistinction to aortic regurgitation, the regurgitant volume may exceed the forward flow volume without there being any elevation of end diastolic pressure in the ventricle.

Pressure curves from the left atrium in both experimental and clinical mitral regurgitation (recorded at operation or by transbronchial puncture or direct puncture from the back) and volume curves recorded with balloons in the esophagus demonstrate a late systolic peak. This may be related to the crescendo type of holosystolic murmur which some patients with MR display.

Several colligative phenomena in mitral regurgitation can be listed: the protodiastolic gallop, rapid outward movement (827) of the

left ventricular wall in early filling (by electrokymography), rapid descent (steep slope) on the limb of the left atrial pressure curve (1172), i.e., that part corresponding to early ventricular filling, rapid inward movement (760) of the left atrial border in the phase corresponding to early ventricular filling (by EKG), the impact on the chest wall which Harvey (652) called "ventricular knock." If the list is to be distinguished from the protodiastolic gallop it should probably be applied to the mechanical phenomenon readily appreciated by palpation.

Any valvular regurgitation represents in essence an internal shunt. Therefore, certain parallels with the hemodynamics and auscultatory findings of the shunts of congenital septal defects are to be expected. For example, mitral regurgitation and ventricular septal defect have points of hemodynamic and clinical (and infra) similarity.

**CARDIOVASCULAR SOUND.** The auscultatory features of mitral regurgitation (Fig. 309) are (1) dull first heart sound at the apex, (2) holosystolic apical murmur, (3) splitting of the second sound at the base, (4) exaggerated third heart sound which may be followed by (5) a short mid diastolic rumble.

Apparently the presence of appreciable mitral leak prevents the sharp tensing of the aortic leaflet of the mitral valve necessary for the production of a first heart sound of normal intensity and sharpness. The first sound may be virtually absent in MR or have only components of low frequency and intensity which endow it with the 'dull' quality as appreciated by the stethoscope. Often the valve in cases of predominant regurgitation is heavily calcified and incapable of much

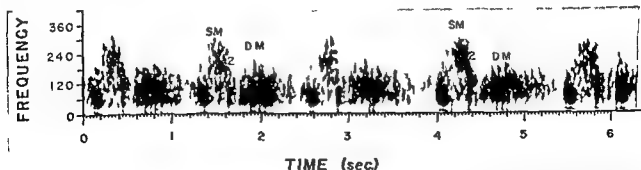


FIG. 309. Predominant mitral regurgitation in L. L. (227489): apex note (1) low frequency content of dull  $S_1$ , (2) relatively high pitch and crescendo character of the systolic murmur, (3) third heart sound initiating diastolic rumble, (4) absence of presystolic murmur because of atrial fibrillation.

relatively high frequency about 800 cps. The result is a murmur to which the analogy of the cooing dove is appropriately applied.

The murmur of ruptured papillary muscle or chordal tendineae (Fig. 317) is usually described as loud, rough, harsh, and coarse (44). Some refer to it as musical, but this is not more often the case; probably because of differing views on what constitutes musicality. Schwartz and Cawley (1332) reported the following investigators' findings in a case of ruptured papillary muscle. A loud harsh musical systolic murmur was heard in the 2nd intercostal space transmitted into the left axilla and posteriorly to the region of the left scapula. The murmur filled the entire period of systole and ended with the second heart sound.

The systolic murmur of aortic regurgitation is well transmitted to the apex and may even confuse it. The phonocardiogram permits differentiation since the murmur of aortic stenosis has a characteristic shape and top before the second sound (p. 268). The systolic murmur of tricuspid regurgitation may be well heard or even loudest at the apex although transmission into the axilla is usually not taking in cases of pure mitral stenosis. Tricuspid regurgitation may create a false impression of accelerated mitral regurgitation.

The second sound at the base is often split in mitral regurgitation (Fig. 318). Early closure of the aortic valve results from the fact that the left ventricle has two periods of discharge. P is less impressive, even exaggerated in mitral regurgitation than in mitral stenosis because pulmonary hypertension is usually less marked.

An exaggerated third heart sound is an important and constant sign of mitral regurgitation. It is the result of increased left ventricular filling in each diastole, the increase involving both volume and rate. Often one feels a sharp impact of the ventricle against the chest wall at the time of the loud third heart sound, a feature which led Harvey to suggest it as the designation of 'ventricular knock' (When the third sound is the loudest of the heart sound (Fig. 319). One must be careful not to confuse the loud third sound for the second heart sound. This is easily done because the second sound is likely to be buried in the first of the murmur.

The third sound may be followed by a short diastolic rumble even though there is little or no mitral obstruction. Rapid and large mitral flow and the dilatation of the left ventricle are responsible.

I frequently take the spectral phonocardiogram demographs in cases of predominant mitral re-

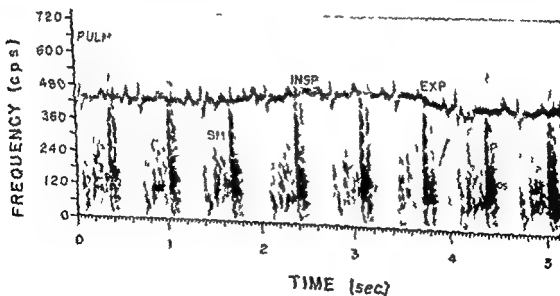


Fig. 317. Split S with mitral regurgitation. 60-year-old female at 60 has an early diastolic murmur at the left (termed) order. The degree of splitting is exaggerated by inspiration.



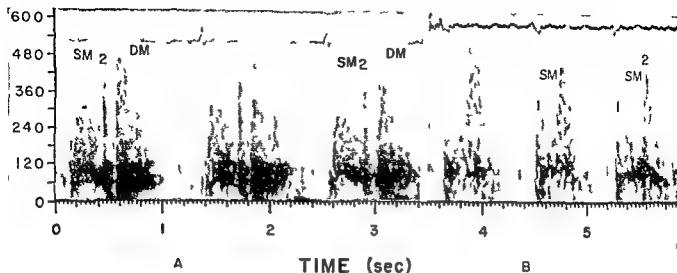


FIG 311 Rheumatic mitral regurgitation

The recording at left was made from a patient who was explored with the intent of performing mitral valvulotomy. Pronounced mitral regurgitation and very little mitral obstruction were discovered. At the apex the first sound is muffled. Possibly two components can be faintly discerned. A decrescendo systolic murmur begins immediately with the second component which is probably a delayed mitral closure sound. The second sound at the apex which probably has its origin largely in closure of the aortic valve is followed by a gap before the beginning of the diastolic rumble. The diastolic rumble begins abruptly with a third sound gallop. No opening snap is demonstrated. The protodiastolic gallop is exceedingly loud, louder in fact than the normal heart sounds.

(At right) the systolic murmur is crescendo in type. Right bundle branch block is present. Splitting of the second sound is barely discernible in some cycles. The murmur continues directly on into early diastole. It is not unexpected that in some anatomic varieties of regurgitant mitral valves regurgitation into the left atrium should continue after closure of the aortic valve and until the time that pressure in the left ventricle has fallen below that in the left atrium. Then a reversal of flow will occur. The net result is that a continuous to and fro murmur occurs; this is rare, however.

posteriorly in the inter-scapular areas and up and down the back, often even over the xiphum (1478)\* or occiput. Furthermore it may be audible to the right of the sternum in the same area in which an expansile systolic pulsation may be seen—about the third right interspace a centimeter or two from the costal margin.

Omundsen *et al* (1170A) and Edwards and Burchell (412) described a case of mitral regurgitation from ruptured posterior chordae tendinae in which aortic stenosis was simulated because of a systolic murmur and thrill to the right of the sternum and even into the carotid arteries. At necropsy a jet lesion of the left atrial endocardium overlying the interatrial septum was found. Movitt and Gerstl (1126A) also had a case of pure mitral regurgitation with systolic murmur in the aortic area and carotids. In another case (274A) of ruptured posterior chordae tendinae a harsh grade IV systolic murmur was loudest at the apex but also loud in the aortic area and

carotids. A misdiagnosis of aortic stenosis was made. The case is less definite because the aortic valve was calcified but not stenosed. Rydmd and Lipsitch (1337) had a case of calcified mitral fibrosis with mitral regurgitation and a systolic murmur well heard in the aortic area and over the carotid arteries.

White (1537) thinks the murmur of mitral regurgitation is louder during heart failure and less intense when myocardial function improves. If the observation can be confirmed by objective measurement it will accord well with the findings of Wiggers and Lail (1557) on the relation between the speed of ventricular contraction and the volume of regurgitation (see above).

The quality of the murmur may be blowing or musical but almost never harsh. A systolic thrill is sometimes felt. A musical murmur is particularly likely to be present during acutely active rheumatic carditis and during and after bacterial endocarditis. The musicality in such cases is usually represented by a single harmonic at

\* See patients D R 9 (A 2/22) and G J (575841)

## VALVULAR HEART DISEASE

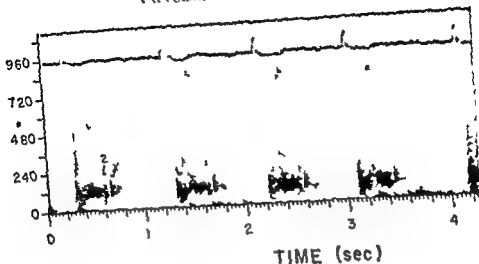


Fig 316 Aortic murmur at apex in 60 year old patient (11-1-15) with superannuated rheumatic heart disease and mitral regurgitation (Note high frequency of the single harmonic)

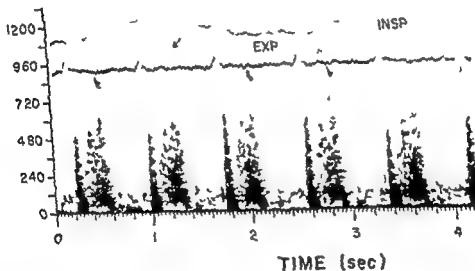


Fig 317 Apex & F (14-1-15) with the Marfan syndrome and aortic mitral regurgitation partly on the basis of ventricular dilatation partly on the basis of redundant heart with weak chordae tendineae. To the ear distinctly musical quality like whistling through a hole in a tube (Note high pitch of single harmonic)

ejection in each case. A holosystolic murmur and by graphic means a mild splitting of the second sound occur in both conditions. In young people with mild aortic regurgitation the murmur may be audible over a large area of the chest and the clinical areas of maximum audibility may not be clear. The presence of a loud third sound gallop may point to mitral regurgitation but is found with ASD as well (Fig 360).

After much discussion and testing of methods for identifying predominant mitral regurgitation

some experienced and perhaps hazardous to the patient many cardiologists have come to the conclusion that aortic regurgitation provides the surest and simplest indicator. It may be appropriate to conclude with a quote from Arthur Ernest Sisson (1849-1907) of the London Hospital writing in his *Valvular Disease* of 1883:

It is only comparatively recently that our pupils could be taught in our hospitals the methods of discriminating

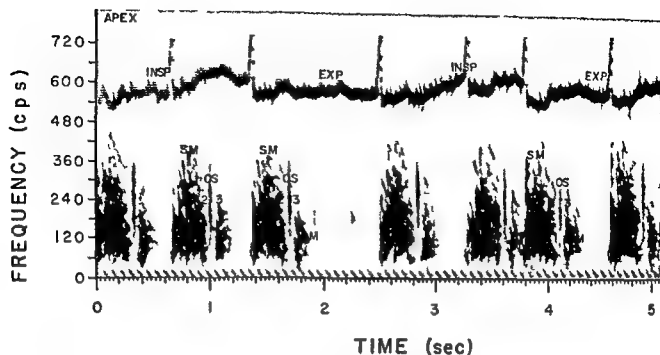


FIG 313 Predominant mitral regurgitation

Apex in M P (767600) 16 year old female with advanced rheumatic heart disease. The clearly demonstrated opening snap does not contradict the diagnosis of predominant mitral regurgitation.

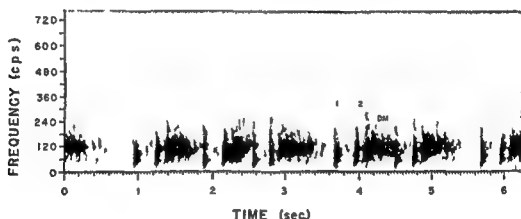


FIG 314 Mitral stenosis with considerable mitral regurgitation atrial fibrillation

gurgitation and diminutive opening snap between the second and third heart sounds (Fig 313).

Simon and Lau (1391) have emphasized the harsh or musical holosystolic apical murmur which may be associated with clefted mitral annulus. Ashworth (34) found in apical systolic murmur in five of ten cases of clefted mitral annulus.

More often than one might perhaps think there is a problem in the differential diagnosis of mitral regurgitation and ventricular septal defect of the Roger type. There are hemodynamic similarities inasmuch as the left ventricle has two ports of

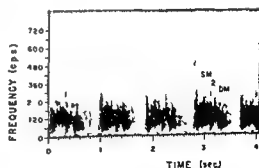


FIG 315 Predominant mitral regurgitation. Essentially continuous murmur. Opening snap situated in interval between  $S_1$  and  $S_2$ ; the latter introduces the main part of the diastolic murmur.

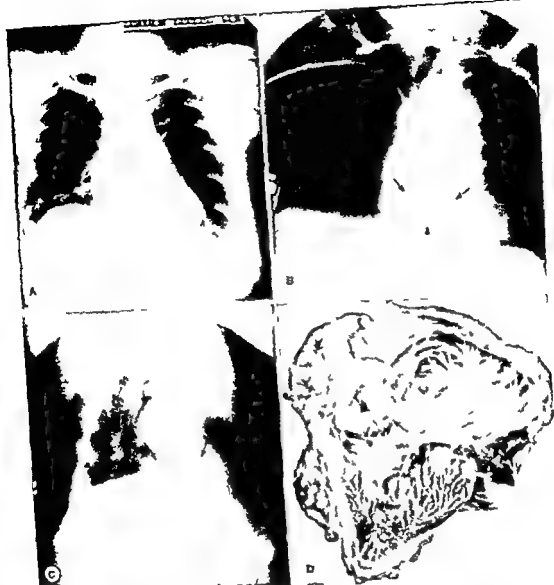


FIG. 320. Myxoma of the right atrium.

B. J. (60384 ant 2391) 61 year old woman half a year before death. History of a gradually increasing pericardial effusion. There is a large tumor (or constrictive pericarditis) associated with a murmur at the left border of the lower sternum. In the conventional film the superior vena cava is dilated and the right atrium bulges to the right. The angiogram (B and C) shows the dilated superior vena cava and in C the enlarged azygos vein. There is a large bill-like filling defect in the right atrium. At autopsy the heart showed remnant of a large pedunculated tumor in the right atrium (D). The greater portion of the tumor had been removed surgically 24 days before death (Case of Babin and Newman (47) illustrations courtesy of C. C. Cole and St. Martin (291)).

are such as the ovary which are drained directly into the venous cavity. Myxoma (Fig. 320) (47) other bill-like neoplasm (Fig. 321) and bill-like thrombus (L.C. 646029) occur on the right side of the circulation as they do on the left with production of a cultory gas

similar to that of rheumatic tricuspid stenosis. Relative tricuspid stenosis with diastolic murmur may occur with high flow and dilated right ventricle in atrial septal defect and anomalous pulmonary venous return. In rare instances relative tricuspid stenosis occurs in case of

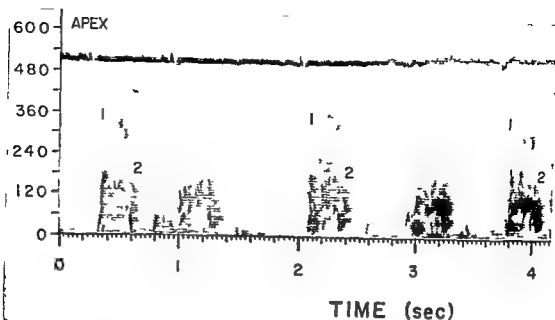


FIG 318 Musical apical systolic murmur in patient (A.C. 580081) with obscure myocarditis and dilated ventricle

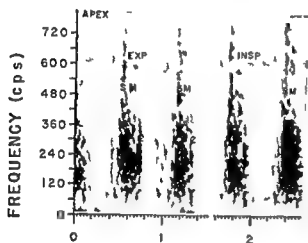


FIG 319 Ruptured chord tendineae

Harsh loud systolic murmur of mitral regurgitation which developed in the 1st year of life in I.D. (690316) 73 years old. There is a faint presystolic crescendo murmur. Autopsy revealed a ruptured chord tendineae. histological clusters of bacteria (cocci) were demonstrated. Other than the appearance of the murmur there were no clinical clues to the diagnosis. In addition to the holosystolic murmur there is both a protodiastolic and a presystolic gallop (indistinctly shown here)

between cases of mitral stenosis and those of mitral regurgitation and it is unwise to conceal the fact that difficulties in such differential diagnosis do occur. I need not ask you to concur with me in deprecating the pleas of *Cui bono?* It is our bounden duty to learn all we can of the disorders we have to treat even if the immediate influence of such knowledge upon treatment be not so very apparent.

### TRICUSPID STENOSIS (TS)

**ETIOLOGIC AND ANATOMIC CONSIDERATIONS** (369)  
A. 712, 1056, 1057) Rheumatic fever is the most frequent cause of this relatively uncommon lesion. Recently, a case of isolated tricuspid stenosis presumably rheumatic has been reported (372). Usually, however, there is involvement of the mitral and aortic valves as well. Gibson and Wood (348) never found tricuspid stenosis with severe mitral regurgitation. They had one case in which lupus erythematosus seemed to be the cause. Congenital tricuspid stenosis (p. 383) is occasionally encountered. Fibroelastosis can be the basis (1218).

Buley and Bolton (43) found significant tricuspid stenosis in 13 of 98 patients with mitral stenosis in which the tricuspid valve also was explored. It is not clear to what extent the incidence in this series was exaggerated by preoperative clinical suspicions of tricuspid stenosis dictating tricuspid exploration.

Because the endocardium of the right atrium is normally thin and the right atrium more distensible than the left, a paper-thin papillary eccentric atrium may occur (1277).

Recently it has become known that by some mechanism scarring occurs in the valves on the right side of the heart when metastasized carcinoid tumors are present in the liver and other





FIG. 321 Occlusive tumor of right atrium

R. B. (545329 aut 22/91) 59 year old man had the clinical picture of constrictive pericarditis. The carcinoma producing the ball valve tumor was of uncertain origin.

pulmonary hypertension apparently on the basis of dilatation of the right ventricle.

**PHYSIOLOGIC CONSIDERATIONS** (457-1600) The most important functional aspect from the point of view of cardiovascular sound is the effect of inspiration on the diastolic murmur. The fall in intrathoracic pressure with inspiration increases venous return ("thoracic aspiration, respiratory pump") and in turn increases the diastolic

murmur of tricuspid stenosis. Killip and Lucas (7924) have demonstrated that the increased tricuspid gradient with inspiration is the result of drop in right ventricular pressure. Pressure in the right ventricle follows intrathoracic pressure closely and drops during inspiration where is any drop in right atrial pressure is cancelled by increased inflow of blood. See Fig. 322A.

The diastolic gradient across the tricuspid

of the murmur in tricuspid stenosis evident in Figures 322 and 323C cannot be taken as an indication that the murmur would impress the ear as being higher pitched. Probably the greater intensity of the component at lower frequencies outweighs the high frequency components. However, Chesterton and Whitaker (208) state that the diastolic murmur may be high pitched and at times have a "egg roll" quality. This I have not observed.

So intense is the diastolic murmur that the unwary can easily misinterpret it as a systolic murmur. Although the diastolic murmur of tricuspid stenosis is usually loudest in the region of the lower left sternal border, one must be prepared to find its point of maximal audibility at least as far to the left as the midclavicular line.

The murmur of relative tricuspid stenosis

heard mainly in cases of atrial septal defect is usually mid-diastolic in timing, blubbery in quality and apical in location. It is not influenced by respiration as a rule.

Relative tricuspid stenosis from dilatation of the right ventricle without necessarily increased tricuspid flow as in ASD has been reported by several writers (see Fig. 439 and p. 432 for such a case)—a patient with primary pulmonary hypertension. In the interesting case of Rivas-Carillo *et al.* (1279) there were both a rumbling, mid-diastolic murmur and a presystolic crescendo. Furthermore, the murmur was accentuated by inspiration as in organic tricuspid stenosis. MacCallum (1003) described a case from the John Hopkins Hospital, a 29-year-old woman (Case 11742) with obliterative lesion of the pulmonary arteries (probably multiple pulmonary

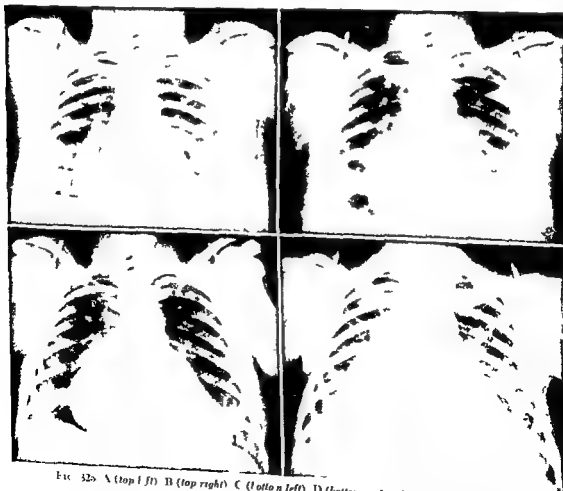


FIG. 325. A (top left), B (top right), C (bottom left), D (bottom right). See legend, Fig. 323.



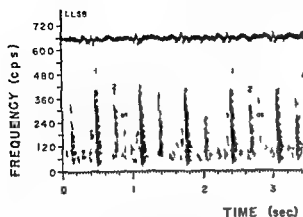


FIG. 323 Tricuspid stenosis

Over lower sternum in J.D. (379600) 22 years old who has had repeated attacks of rheumatic fever. There are signs of both aortic and mitral involvement but those of tricuspid disease are especially striking: giant waves in the jugular pulse and presystolic murmur with thrill over the lower sternum accentuated by or present only during inspiration. Large right atrium. The presystolic murmur was accentuated by inspiration and lightly separated from  $S_1$ . The frequency span of the presystolic murmur was greater than is usually seen in cases of MS. The opening snap is probably tricuspid; it shows a greater  $S_1$ -OS interval than in the case of mitral OS and in recordings taken with breathing is virtually absent in expiration.

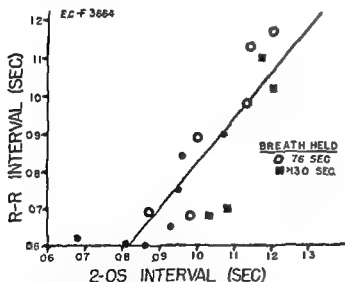


FIG. 324 Tricuspid stenosis—interval between  $S_2$  and tricuspid opening snap. All measurements from one recording. Atrial fibrillation. Holding the breath had little effect. (From Kossmann (816).)

sound. In fact the first sound has been essentially absent over the lower sternum in some cases (e.g. Fig. 322). The tricuspid opening snap is rarely as close to the second sound as is its

mitral counterpart. Mainly this is because pressure in the right atrium rarely attains as high a level as in the left atrium, probably because the systemic venous compartment is so much larger than the pulmonary venous compartment. The right atrium is more distensible than the left has different pressure-volume characteristics. In small part, the greater delay may be apparent rather than real, one must relate the tricuspid opening snap to the later pulmonary component of the second sound, not to the aortic component. The  $S_2$  to tricuspid OS interval is likely to vary with respiration, being shortest with inspiration for reasons identical to those for the exaggerated diastolic murmur with inspiration.

The diastolic murmur of tricuspid stenosis has the same two components—early mitral and late aortic—as that of mitral stenosis. It differs from the murmur of mitral stenosis in several respects: (1) It is likely to show impressive variability with phase of respiration, being loudest in late inspiration or in the period of so-called inspiratory pause (held inspiration). This phenomenon is sometimes called Carvallo's sign (1276, 1277). As a rule the murmur of mitral stenosis shows little respiratory variation if it were at all it is likely to be loudest in expiration. However, I have encountered at least two instances of diastolic murmurs clearly of mitral origin, which were accentuated on inspiration. Similarly, Schilder and Harvey (1357) noted that although the murmur of mitral regurgitation is likely to decrease with inspiration both the opening snap and the diastolic rumble of mitral stenosis may be increased. They state that a number of patients have been observed who demonstrated this. Furthermore the murmur of tricuspid stenosis of most severe degree is uninfluenced by respiration. Apparently venous pressure is already so elevated that the increment produced by inspiration is inconsequential in its effects.

(2) The murmur of tricuspid stenosis tends to be louder, to be accompanied by a striking thrill and to have a higher frequency span in the spectral phonocardiogram than the murmur of mitral stenosis. The reason for these three intimately related phenomena is probably the more superficial position of the generator area in the case of the tricuspid valve. The greater frequency range

of the murmur in tricuspid stenosis evident in Figure 22 and 23f cannot be taken as an indication that the murmur would impress the ear as being higher pitched. Probably the greater intensity of the component at lower frequencies outweighs the high frequency component. However, Chesterman and Whitaker (238) state that the diastolic murmur may be high pitched and at times have a "ex gull" quality. This I have not observed.

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Relative tricuspid stenosis from dilatation of the right ventricle without necessarily increased tricuspid flow as in ASD has been reported by several writers (see Fig. 139 and p. 432 for such a case)—a patient with primary pulmonary hypertension. In the interesting case of Rivero Carillo et al. (1279) there were both a rumbling mid-diastolic murmur and a presystolic crescendo. Furthermore, the murmur was accentuated by inspiration as in organic tricuspid stenosis. MacCallum (1004) described a case from the Johns Hopkins Hospital, a 29-year-old woman (83 out 11742) with obstructive lesion of the pulmonary arteries (probably multiple pulmonary

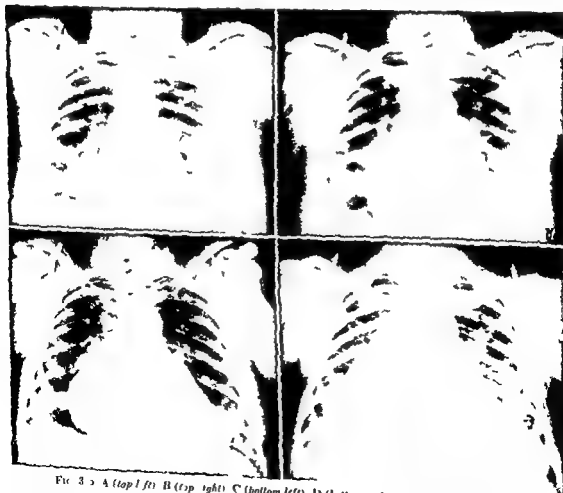


FIG. 3—A (top left) B (top right) C (bottom left) D (bottom right) (see legend Fig. 3-1)

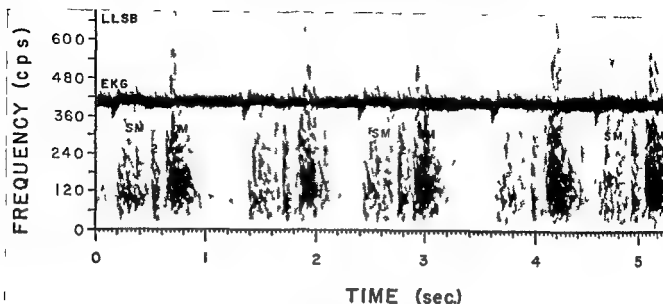


FIG 325 E Tricuspid stenosis

C D (127526) 41 year old female has been seen at this hospital since 1933 when a chest x ray already showed fullness of the right atrium consistent with tricuspid disease (1) This showed steady increase in the films taken in 1948 (B) 1952 (C) and 1957 (D) There is atrial fibrillation enormous distension of the neck veins and liver a striking diastolic rumble accompanied by thrill in the left midprecordium E presents sounds at LLSB Diastolic being this murmur from that of mitral stenosis the large frequency span As in the case displayed in Figure 377 the first heart sound is faint

emboli) and a mid diastolic rumble at the apex. In 1935 Wyckoff and Bunim (1595) described three such cases and found ten in the literature. One of their cases was a 21 year old Puerto Rican woman seen in New York with cor pulmonale due to *Schistosoma mansoni*. They emphasized that the diastolic rumble could be quite constant, it was heard consistently over a period of seven years.

White (1630) wrote in 1947: "Functional tricuspid stenosis has not been described although I have recently encountered two cases which I believe to be such with well localized mid diastolic murmurs near the lower end of the sternum, mitral diastolic murmurs and loud pulmonary diastolic murmur." He was apparently discussing cases of rheumatic heart disease. It is entirely possible that some of the instances of what has been interpreted as organic tricuspid stenosis with mitral stenosis have largely or only relative tricuspid stenosis. Reubi, Vogt and Plancherel (1263) described the case of a patient with pericarditis and pulmonary hypertension who had signs of pulmonary regurgitation and right sided hypertrophy. In addition there was at the apex a diastolic murmur suggesting mitral stenosis but interpreted by the authors

as the murmur of relative tricuspid stenosis. Davis and Andrus (316) described a case of mediastinal collagenosis in which the pulmonary veins were severely constricted and a rumbling apical diastolic murmur produced probably through the mechanism of relative tricuspid stenosis. Bruchfeld *et al* (147A) described two patients with primary pulmonary hypertension and a diastolic rumble of relative tricuspid stenosis.

A right sided Austin Flint murmur may occur in cases of pulmonary hypertension with pulmonary regurgitation. I have observed three such instances. These are cases in which a conspicuous pre-systolic concentration of the diastolic murmur (of pulmonary regurgitation) at the lower left sternal border is demonstrated.

A sound suspiciously suggestive of an opening snap is even seen in some cases of the same type and may have the murmur of relative tricuspid stenosis although not necessarily in association with a murmur.

#### TRICUSPID REGURGITATION (TR)

(Syn Tricuspid insufficiency or incompetence)

ETIOLOGIC, ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS (905-1133) An organic cuspal basis

is rather uncommon being of rheumatic or congenital origin when it occurs. Relative tricuspid insufficiency on bases similar to those outlined for the mitral valve (p. 314) e.g. right ventricular failure (813/64) is common. Morphine addicts ('mainliners') are prone to bacterial endocarditis affecting the tricuspid valve.

The anatomic and physiologic changes are similar to those for the mitral valve. Relative insufficiency occurs more readily than in the case of the mitral valve.

**CARDIOVASCULAR SOUND** The systolic murmur of tricuspid regurgitation differs from that of mitral regurgitation mainly in its location of maximum audibility and in its change with respiration. Both however are not invariable features distinguishing this lesion from mitral stenosis.

The holosystolic murmur of tricuspid regurgitation is usually loudest in the left midprecordium. Purely aortic in the tricuspid area. Dilatation of the right ventricle which occurs either as the cause or the result of the tricuspid regurgitation brings the right ventricle unusually far to the left. Often in cases of mitral stenosis a loud systolic murmur at the apex is attributed to the association of insignificant mitral regurgitation. However if urgency performed it may be found that there is no regurgitant jet. Furthermore with

urgent relief of the mitral obstruction the systolic murmur may disappear. This sequence of events suggests that right-sided heart failure with relative tricuspid insufficiency produced the systolic murmur well heard at the apex and falsely interpreted as being caused by mitral regurgitation (137/1482). With the onset of atrial fibrillation the evidence of tricuspid regurgitation in association with right ventricular failure may be especially striking. In one 33-year-old patient (133/703881) who had giant aneurysm in the venous pulse and a presystolic pulsation of the liver as well as an oscillatory sign of tricuspid stenosis but no evidence of tricuspid regurgitation striking systolic pulsation of the liver appeared in the 11th week of life with the development of atrial fibrillation and pronounced heart failure.

The systolic murmur of TR may be accentuated by inspiration (1278) through an increase in venous return and therefore in right ventricular stroke volume. However as compared with tricuspid stenosis the influence of respiration on the murmur of tricuspid regurgitation is less impressive. Furthermore with right ventricular failure which is usually present the ventricle is already operating at maximum capacity and cannot increase its cardiac output appreciably.

In severe tricuspid regurgitation a loud murmur related to ventricular systole may be strikingly

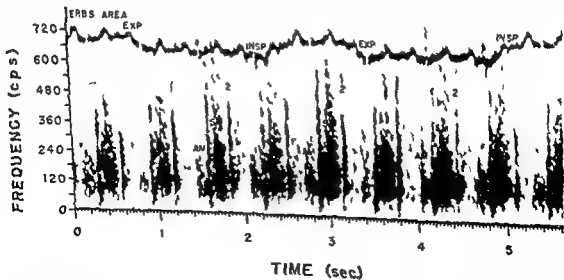


FIG. 378. Tricuspid stenosis. A 17-year-old female has typical murmurs of AS and AR. Tricuspid stenosis is indicated by the prominence of the presystolic murmur at Erb's area (shown here) its early occurrence with slight separation from the normal PR interval and its exaggeration with inspiration.

audible over the veins of the extremities I am told (840) of an instance in which the murmur was so impressive in the region of the femoral triangle that, in combination with the tumescence of engorged veins in that area, arteriovenous fistula was misdiagnosed.

### PULMONARY STENOSIS (PS)

Rheumatism affects the pulmonary valve very rarely. The overwhelming majority of cases of PS have a congenital basis. However, as part of circumoid cardiovascular disease, there may be acquired PS, combined usually with pulmonary regurgitation and with both types of functional defect at the tricuspid valve as well.

Relative PS is frequent. The most striking example is atrial septal defect in which there may not only be a striking systolic murmur but also a pressure drop of as much as 40 mm across the pulmonary valve. Voluminous right ventricular outflow and dilation of the outflow tract above and below the valve are responsible. Just as in the case of aortic stenosis the differentiation of marked relative from mild organic pulmonary stenosis may be difficult.

So rare is any variety of PS except congenital that reference is made to the discussion of congenital pulmonary stenosis on pp 365-375.

### PULMONARY REGURGITATION

**ANATOMIC ETIOLOGY AND PHYSIOLOGIC CONSIDERATIONS.** Relative pulmonary insufficiency secondary to pulmonary hypertension with production of the Graham Steell murmur is not uncommon (99). Actual rheumatic damage to the pulmonary cusps is rare. The gonococcus has a notorious propensity to localization on the pulmonic valve; at least the gonococcus attacks the pulmonic valve more frequently than most other organisms although when all cases of gonococcal endocarditis are analyzed involvement of the valves on the left side of the heart occurs more commonly than pulmonic involvement. Pulmonary regurgitation along with other valvular defects can occur in circumoid cardiovascular disease. Trauma may result in pulmonary regurgitation. Having enumerated these conditions all rule except for the Graham Steell

murmur, the acquired types of pulmonary regurgitation have been rarely, if not completely, exhausted.

Isolated pulmonary regurgitation on a congenital basis is likewise uncommon. As was known to Leonardo, a quadricuspid semilunar valve is from the engineering standpoint, not as strong as a tricuspid valve. Pulmonary regurgitation has been reported with quadricuspid valve (798) and with bicuspid valve (352A).

Pulmonary regurgitation relative or organic occurs with congenital malformations such as pulmonary stenosis, Eisenmenger complex, ventricular septal defect, idiopathic dilatation of the pulmonary artery, etc. Isolated pulmonary regurgitation presumably congenital has been diagnosed in life by clinical and physiologic means by Kjellberg *et al* (800) Lord (469) and Morton and Stern (1117). Absence of the pulmonary valve with pulmonary regurgitation has been reported (229); the patient also had a ventricular septal defect. The pulmonary valve may be absent in cases of tetralogy of Fallot with infundibular stenosis.

With pronounced pulmonary regurgitation there is likely to be a striking hollowness on fluoroscopy and by electrokymograms or roentgen kymograms. Large excursions of border movement in the region of the main pulmonary artery and rapid rise and fall of the limbs of the curves. By cardiac catheterization there is usually a wide pulmonary arterial pulse pressure. Particularly diagnostic is the steep slope of the diastolic limb with fall of pressure in diastole to a level almost equal that in the right ventricle (22, 1117).

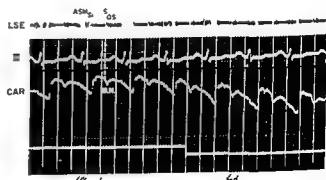


FIG 327. Improved tricuspid stenosis. Note increase in pre-systolic murmur (ASM) with inspiration. S<sub>1</sub> = first sound & = second sound OS = opening snap (from Harvey (652)).

Ellison and colleagues (423) found that the ejection volume was increased sufficiently in their dogs to produce a systolic gradient of 1 mmHg or much as 40 mm Hg across the valve orifice in the absence of organic obstruction. The same gradient has been found in man (22, 409).

Pulmonary regurgitation is a well tolerated valve lesion. Burger and colleagues (424) and Ellison and colleagues (425) could not produce heart failure in dogs by destruction of the pulmonary valve. On the other hand, Kay and Thomas (773) had one dog that died of right-sided heart failure and thought that others would have done so if exercised. They attributed the difference in results to more complete excision of the pulmonary cup. Oleson and Liberman (1152) found that a patient had little incapacitation from pulmonary regurgitation of severe proportion 27 years after gonococcal endocarditis. Campbell and colleagues (229) describe a 32-year-old with no pulmonary valve and death which was adequately accounted for by the associated lesion—A-V-D and high coronary artery with myocardial infarction.

**CARDIOVASCULAR** The Graham Steell murmur because of the high pressure in the pulmonary artery often mimicking or exceeding that in the aorta is high pitched and resembles the murmur of aortic regurgitation precisely. It may be accentuated by inspiration (1153, 196641)—a feature which does not occur often enough to be too helpful.

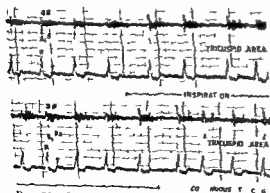


FIG. 375. Murmur of aortic regurgitation exaggerated by inspiration. This patient has surgically proved pure mitral stenosis. The systolic murmur could easily have been misinterpreted as indicating mitral regurgitation (Cf. note of Harvey, 1677) and (Calculation).

In organic pulmonary regurgitation the diagnosis may be suggested by the fact that although the murmur begins immediately with the second heart sound it is relatively low pitched. When the murmur is generated at normal level of pulmonary artery pressure (about 20 mm Hg at the time of pulmonary artery closure) a murmur of quite different quality is produced, as compared with the situation when there is pulmonary hypertension.

Hollick and his colleagues (704, 1102) were impressed with the late onset or crescendo-decrescendo pattern of the diastolic murmur of pulmonary regurgitation. They describe with phonocardiogram cases of pulmonary regurgitation and intervals of 0.05 to 0.10 sec between the second sound and the onset of the murmur. In experimentally produced pulmonary regurgitation in dogs, Rogers and his colleagues (1457) found a crescendo-decrescendo pattern to the diastolic murmur or a gap between the second sound and the onset of the diastolic murmur.

In a patient (C.S.M. 24529) who appears to have ventricular septal defect with pulmonary hypertension the only murmur is a low, rough diastolic murmur and thrill produced probably by pulmonary regurgitation. It is non-decrescendo. Systolic and diastolic have been of approximately equal length. Experienced observers have repeatedly mistaken it for a systolic murmur. Hollick and colleagues (702) described a very similar murmur in a patient with patent ductus arteriosus and pulmonary hypertension.

Branden (1305) described a holodiastolic murmur with atrial septal defect and pulmonary hypertension. A diastolic thrill was palpable in one case.

#### LESS COMMON VARIANTS OF ACQUIRED VALVULAR HEART DISEASE

Bacterial endocarditis will be discussed later (pp. 430-432).

In gout tophi may occur on the mitral valve. The most acceptable case is that of Bunton and McEwen (190). The posterior mitral leaflet was the site of a large urate tophus. In life no murmur was heard. This is probably not surprising since the anterior (or aortic) leaflet alone is often adequate for closing the mitral orifice. Furthermore the tophus was located in the belly rather

than at the margin of this leaflet I have observed a 52 year old patient (P L, 692200) with extremely severe tophaceous gout and a loud mitral systolic murmur of obscure cause

In *ochronosis* (*alkaptonuria*), there may be deposit of the anomalous material not only in the intervertebral disks, leading to calcification and ankylosing spondylitis, but also in the heart valves, with production of calcific aortic stenosis

(945A) In one case the authors stated "The aortic valve was stenotic and also showed extensive pigmentation and nodular calcification" (945A)

With *carcinoid tumors* (*argentaffinomas*) of the small bowel, especially with metastases in the liver or other sites, such as the ovary, which have direct venous drainage to the right side of the heart, there occurs damage to the tricuspid and



FIG 329 Appearance of patient described by Sir Maurice Cady (204) probably the first reported instance of *carcinoid cardiovascular disease*. The flushing and the telangiectases in the butterfly areas of the face are well shown. The original in color well demonstrates patchy cyanosis over the upper part of the chest.

pulmonary valves with stenosis and regurgitation (Figs. 329 to 332). The precise mechanism is not clear. However, something elaborated by the tumor and partially destroyed in the liver and lung seem to be responsible. In one of the cases reported (1068) the foramen ovale had been secondarily opened as a result of pulmonary hypertension, the right-sided valve lesion, and right-sided failure. This patient developed not only polycythemia but also changes in the mitral valve and to some extent the aortic.

A few instances of mitral stenosis in association with giant cell aortitis of the temporal arteritis type have been described (1252). The significance of the association is unclear.

With impressive frequency, aortic regurgitation is associated with Marie-Strumpell *spondylitis*. Schilder and colleagues (1758) found *spondylitis* in five cases of aortic regurgitation. Since

*spondylitis* occurs with overwhelming preponderance in males, one should consider particularly the incidence of *spondylitis* in male patients with aortic regurgitation. 83 per cent. The regurgitation was even leading to death in three patients during the period of observation. Histologically there was extensive change in all three layers of the base of the aorta. Blumberg and Riggs (121) found six cases of valvular heart disease among 128 cases of rheumatoid *spondylitis*. Changes in the heart valves, especially in the mitral and usually located midway between the margin and base, are found commonly in cases of peripheral rheumatoid arthritis, which come to autopsy. The changes resemble those of the rheumatoid nodule both grossly and microscopically. Usually the lesions are of limited functional significance. However, Bugga toos and Rosenberg (41) in a report which is one of the earliest on the subject



FIG. 330 Liver metastases in a case with carcinoid carcinoma of the lung.  $\times 100$





FIG. 331 The gross appearance of the heart valves in a patient with carcinoid cardiovascular disease. (Upper left) Tricuspid valve. Note the sclerosis of the endocardium of the right atrium and perforation of the interatrial septum. (Upper right) Outflow tract of right ventricle with sclerotic pulmonary artery above, sclerotic tricuspid valve below. (Lower left) Close up of sclerotic pulmonary valve. (Lower right) Sclerotic chordae tendineae of mitral valve. Sclerotic of the mitral and aortic valves occurred in this case probably because of passage of blood to the left side via the atrial septal defect without circulation through the lung.

of the heart in rheumatoid arthritis described a case of tight mitral stenosis with classical deforming rheumatoid arthritis. Bauer and colleagues (62) have written about the aortitis and aortic valve disease of rheumatoid arthritis. Clark, Kulka and Bauer (271) found valvular involvement in 4.8 per cent of 1000 patients with rheumatoid arthritis—2.2 per cent had aortic regurgitation alone, 1.4 per cent had aortic regurgitation in combination with mitral stenosis.

aortic regurgitation alone or in combination with another valve lesion was present in the entire 48 patients. The patients with valvular involvement were almost exclusively men. Involvement of the spine was present in 90 per cent and of the hands in 50 per cent. The incidence of uveitis (60 per cent) was higher than in rheumatoid arthritis in general. Angina pectoris presumably from involvement of the coronary ostia is part of the rheumatoid aortitis, was frequent. In this respect

and others—such as the long course with rapid deterioration once failure sets in—close simulation of syphilitic heart disease is produced

Figure 33 shows the main autopsy finding (autopsy 21422) in a child with tuberous sclerosis. A rhabdomyoma bulged into the outflow tract of the right ventricle producing a loud systolic murmur at the left sternal border and right-sided heart failure. A similar situation resulting from a fibroma of the left ventricle has been described (1039) Although these are not strictly valvular the murmurs and the pathologic physiology suggest a valvular defect

In diffuse systemic sclerosis (systemic sclerosis) there may be a bilateral diastolic murmur (eg I S 72932) There are two possible mechanisms (1) pulmonary regurgitation from fibrosis of the lung and pulmonary hypertension (2) aortic regurgitation from a lesion of the valve

In fusion of the dilated aortic root into the mitral orifice (1573) in hypertension a phloidesecting aneurysm (504) or the Marfan syndrome is a theoretically possible cause of mitral diastolic murmur

Valvular involvement in some degree occurs in about 40 per cent of cases of primary systemic amyloidosis (1321) Occasionally (766 3022 3177) it is severe enough to produce important functional defect In many other cases murmurs both systolic and diastolic occur

Quercetium of the mitral valve Hennrich and Stewart (1261) describe a 63-year-old patient who in the last year of life went blind from endocarditis developed a loud blowing mitral systolic murmur and went into congestive heart failure At autopsy the anterior mitral leaflet was distorted by a pouch large enough to accommodate the tip of the little finger to a depth of over 1 cm The pouch was directed backward so that during ventricular systole it would impinge on the posterior cup of a rounded hill having a triangular orifice on either side The pathogenesis is unknown

In systemic lupus erythematosus (6382) I have had an atypical aneurysmal region of pure mitral stenosis in one case (A 44337) and of mitral stenosis and regurgitation and aortic regurgitation in another (B T 429876) In a third patient (C A 317313) there is profound

aortic regurgitation which in large part had its basis in two bouts of bacterial endocarditis complicating Libman-Sacks lesion of the aortic valve Figure 33 presents a case of mitral stenosis due possibly to SLE In 1921 in the classic article on a hitherto undescribed form of valvular and mural endocarditis Libman and Sacks (913) referred to the presence of a gross tricuspid regurgitation in three cases In one which came to post mortem examination thickening of the mitral valve but no chord bodies were found (1573) and Ward (215) found typical rheumatic mitral stenosis in a patient whose other clinical and pathologic features were those of lupus Shearn and Firokz (1581) found a systolic murmur in 21 of 33 cases, a diastolic in 7 of 33 and in apical diastolic murmur in 15 of 33 The diastolic murmur was presystolic in one and mid-diastolic in three Both patients who came to post mortem examination displayed Libman-Sacks changes of the mitral valve (Shearn and Ward (158) described the case of a 33-year-old man with presumed SLE and presumed isolated tricuspid stenosis All clinical findings were consistent with these diagnoses but histology were not identified The valvular involvement in SLE is more impressive pathologically than clinically Involvement of the pulmonary and tricuspid valves is often more striking than that of their counterpart on the left side Clinically an apical systolic murmur occurs in possibly half of the cases Collapsus occurs frequently Hypertension and its electrolyte changes are usually absent except with end-stage renal involvement and/or with steroid therapy

Libman-Sacks is important in the pathology of the aortic valve frequently involves the ventricular aspect of the aortic cusset of the mitral valves in a plaque distribution (11 661 311) Rarely if ever is this involvement per se of hemodynamic clinical or some significance (158)

With calcification of the annulus fibrosus mitralis a high systolic murmur may develop (1121) The murmur probably is on the basis of mitral regurgitation This is a variety of relative insufficiency interference with contraction of muscular component of the mitral ring probably responsible It must be pointed out that Firtman and Wolff (438) were unable to relate the occurrence of a murmur to calcification of the mitral annulus



FIG. 331 The gross appearance of the heart valves in a patient with *carcinoid cardiovascular disease* (Upper left) Tricuspid valve. Note the sclerolysis of the endocardium of the right atrium and perforation of the interatrial septum (Upper right) Outflow tract of right ventricle with curved pulmonary artery above, curved tricuspid valve below (Lower left) Close up of scarred pulmonary valve (Lower right) Beirred chordal tendineae of mitral valve. Scarring of the mitral and aortic valve occurred in this case probably because of passage of blood to the left side via the atrial septal defect without circulation through the lungs.

of the heart in rheumatoid arthritis described a case of tight mitral stenosis with classical deforming rheumatoid arthritis. Bauer and colleagues (62) have written about the aortitis and aortic valve disease of rheumatoid arthritis. Clark, Kulkarni and Bauer (271) found valvular involvement in 48 per cent of 1000 patients with rheumatoid arthritis—22 per cent had aortic regurgitation alone, 14 per cent had aortic regurgitation in combination with mitral stenosis.

aortic regurgitation alone or in combination with another valve lesion was present in the entire 48 patients. The patients with valvular involvement were almost exclusively men. Involvement of the spine was present in 90 per cent and of the hands in 50 per cent. The incidence of uveitis (60 per cent) was higher than in rheumatoid arthritis in general. Angina pectoris, presumably from involvement of the coronary ostia as part of the rheumatoid aortitis, was frequent. In this respect



FIG. 337 H

FIG. 337. Carcinoid endocardial disease. Microscopic appearance of the valves in the same case as illustrated in the last figure. A. Tricuspid valve  $\times 4$ . B. Pulmonary valve  $\times 4$ . C. Mitral valve  $\times 4$ . D. A mitral chorda tendinea  $\times 8$ . E. Aortic cusp  $\times 23$ . F. Normal aortic cusp from a heart for comparison  $\times 23$ . G. Bulbous tip of tricuspid valve. Ventricular surface above; atrial aspect below. Hyaline avascular and acellular material is present on the atrial side of the valve  $\times 75$ . H. Striking evidence of current activity of inflammatory process  $\times 100$ .



FIG. 338. Aneurysm in outflow tract of right ventricle. Loud systolic murmur. The child had tuberous sclerosis.



FIG 332 A (left) and B (right)



FIG 332 C (left) and D (right)



FIG 332 E (left) and F (right)



FIG 332 G



FIG. 332 H

FIG. 332 Carcinoid cardiovascular disease. Microscopic appearance of the valves in the same case as illustrated in the last figure. A Tricuspid valve  $\times 4$  B Pulmonary valve  $\times 4$  C Mitral valve  $\times 4$  D A mitral chorda tendinea  $\times 8$  E Aortic cu p  $\times 23$  F Normal aortic cu p from adult for comparison  $\times 23$  G Bulbous tip of tricuspid valve. Ventricular surface above atrial aspect below. Hyaline avascular and acellular material is present on the atrial side of the valve  $\times 30$  H Striking evidence of current activity of inflammatory process  $\times 100$



FIG. 333 *Trichinella* in outflow tract of right ventricle. loud systolic murmur. This child had tuberous sclerosis.

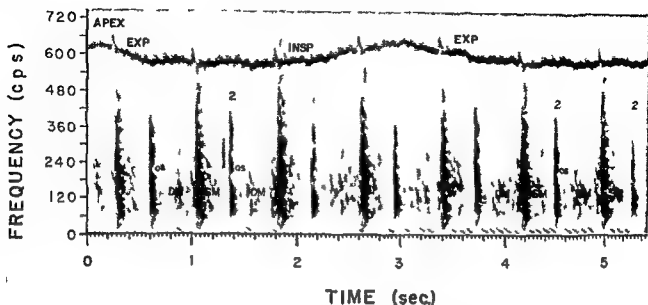


FIG. 334 Probable mitral stenosis in patient with chronic polyarthritis possibly SLE

I. B. T. (32084) now age 43 had a butterfly rash and a chronic illness manifested by repeated rapid accumulations of peritoneal and pleural fluid requiring frequent paracentesis. In recent times fluid accumulation has been controlled by salt restriction and mercurial diuretics. However no culatory sign of mitral stenosis have appeared. On cardiac catheterization performed at the time of this recording the pulmonary capillary pressure at rest was normal. The recording shows undoubted signs of mitral stenosis snapping  $M_1$  opening snap diastolic rumble with presystolic crescendo  $M_1$  is delayed very little.

Blackman (110) reported on *syphilis of the mitral valve* resulting in mitral regurgitation.

With *endomyocardial fibrosis* (not to be confused with fetal fibroelastosis) the valve mechanism—in the case of the tricuspid or the mitral or both—often is so affected that a systolic murmur which is often high pitched and may be accompanied by a thrill, is produced (332). Adhesion of the posterior mitral cusp with immobilization is a frequent basis of mitral regurgitation. This disease occurs with extraordinarily high frequency in the natives of some parts of Africa (47). Diastolic murmurs were unimpressive both in incidence and intensity in the 20 cases reported by Bill Davies and Williams (47) from Uganda.

During *insulin shock therapy* a wide pulse pressure is likely to be observed and a bisular diastolic murmur may be heard. Although I have

not studied the matter systematically, I am told by a keen observer of a patient (J. K., 710776) who with insulin administration developed 'acute aortic insufficiency' with 'zero diastolic pressure' and an aortic diastolic murmur. With glucose administration both of these manifestations disappeared in a few minutes. Melko and Holldick (1102) have made similar observations. They claim to reproduce the entire picture, including the bisular diastolic murmur, with epinephrine administration and suggest that this is the mechanism of the phenomenon with insulin shock. They further suggest that the pulmonary valve is the source of the murmur mainly because the murmur displayed characteristics they ascribe to that of pulmonary regurgitation—crescendo-decrescendo pattern or more often, a brief gap between S<sub>2</sub> and the onset of the murmur.

## CHAPTER 16

# Congenital Cardiovascular Disease

The order in which the various congenital malformations will be discussed has been chosen not on the basis of any complicated embryologic or other system of classification but rather with a mind to eliminating repetition as much as possible.

### ATRIAL SEPTAL DEFECT (ASD)

(Syn: Interatrial septal defect (IASD)  
interatrial septal defect patent  
foramen ovale (etc.))

**DEFINITION** Any permanent patency of a portion of the interatrial septum.

**ANATOMIC CONSIDERATIONS** (719) The valve of mere flap patency of the foramen ovale which is present in about 16 per cent of cases (719) and will not concern us here; two anatomical types are recognized: (1) Atrial septal defects of the patent foramen secundum type are located relatively high in the septum in the area of the fossa ovalis and there is usually no involvement of the atrioventricular valves. The secundum type of atrial septal defect is anatomically hemodynamically and clinically the classical one and the one most amenable to presently available method for surgical repair. (2) With persistence of the foramen primum the defect is located low in the atrial septum close to the atrioventricular ring, and abnormality of the tricuspid or mitral valve with regurgitation at one of these valves is likely to occur. The second type constituting 10 to 15 per cent of all cases of ASD (228) is a variety of endocardial cushion defect (see p. 350).

Frequently, instead of septal remnants persisting, the septal defect (Fig. 33) (ASD Figs. 1, 2 and H 44-49).

Left to right shunt (1) usually all cases of atrial septal defect.

The other anatomical findings are consequences of the shunt. The right atrium, ventricle and pulmonary artery are usually greatly dilated. Hypertrophy also occurs. The enlargement of the right side of the heart tends to produce rotation of the heart in a clockwise direction (viewing the heart from the apex). The right ventricle comes to occupy the cardiac apex and to extend further to the left than is ordinarily the case. The left atrium, left ventricle and aorta are usually small.

Lembert's syndrome (907, 1061)—ASD with mitral stenosis (Fig. 33)—is rarer than is often thought. Among 2,000 autopsies (1138) there were 87 cases of ASD of which 5 had associated mitral stenosis.

**PHYSIOLOGIC CONSIDERATIONS** In uncomplicated ASD the net shunt is from the left atrium to the right atrium. It is necessary to peak off the net shunt because studies—particularly with injection of dye into a vein of a lower extremity—demonstrate that there is often a small right to left shunt involving inferior vena caval blood. Thus right to left shunt usually is not sufficient to produce a degree of oxygen unsaturation of arterial blood below what is accepted as the lower limit of the normal range.

The magnitude of the shunt may be such that the right ventricle pumps considerably more blood than the left ventricle. Lemberton (1199) found as have others that systemic flow is normal in most cases of ASD. In five cases pulmonary flow exceeded systemic flow by 36 to 78 per cent (average 60 per cent); others have found pulmonary flows more than twice normal. When the volume of the shunt is very large the arteriovenous oxygen difference becomes so small as to fall within the range of error for the method. A small deviation in the determination of pulmonary





FIG 335 Remnant of atrial septum in case of large secundum defect. Viewed from left side in V B (406046) 33 year old white female with Lutenbacher's syndrome. The mitral stenosis was rheumatic. The scarred mitral valve is visible in the lower portion of the photograph

artery oxygen saturation results in a large difference in the flow value calculated.

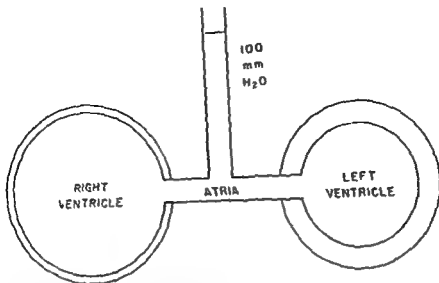
In the past, various theories have been advanced to account for the left to right shunt. (1) Normally, the pressure in the left atrium is slightly higher than that in the right atrium. However, with an ASD of clinically significant size the two atria are fundamentally one chamber and by the laws of physics pressure must be identical in all parts of this chamber. (2) Since the left atrium usually is superiorly located in relation to the right atrium, it has been suggested (1480) that by gravity blood tends to fall into the right atrium from the left atrium. This suggestion was put to critical test by Brannon, Weems and Warren (160), who could demonstrate no change in the direction or degree of the shunt with changes in position.

(3) The most credible possibility is that the left to right shunt is related to the difference in pressure-volume characteristics of the two ventricles (351, 369, 723, 1029). The concept can be represented by the diagram in Figure 336. The two ventricles are represented as balloons, one (the "right ventricle") thin walled and one (the "left ventricle") thick walled, connected to a common reservoir and pressure head, represented as a T tube. Because of the less steep pressure-volume curve of the right ventricle,<sup>2</sup> an expression of its greater distensibility, the right ventricle accepts the larger volume of blood from the common reservoir, as would be predicted from the simplified model with balloons (Fig 336). In accordance with this concept, it is significant that the left to right shunt occurs mainly during ventricular diastole. Therefore, the systolic murmur is not likely to be generated at the septal defect itself.<sup>3</sup> This theory does not exclude the possibility that some blood may be transferred from the left atrium to the right atrium even during ventricular systole. In fact this seems very likely for at least two reasons: (1) the right atrium is more distensible than the left (Fig 337), (957), and (2) the right atrium becomes large in ASD. The greater distensibility of the right atrium is predictable from the difference in the endocardial lining. That of the left atrium is fairly fibrous, giving a relatively smooth and pearly gross appearance. That of the right atrium is thin so that the muscular trabeculations are clearly seen.

To electrocardiographers and particularly in congenital heart disease the concept of systolic vs diastolic ventricular overload (210-211) is useful (1602) since each variety is usually attended by a distinctive electrocardiographic pattern. In conditions of systolic overload of the right ventricle exemplified by pulmonary stenosis, there are simple evidences of ventricular hypertrophy: right axis deviation in the standard limb leads and R waves of large amplitude in leads at right end of the precordial series. In conditions

<sup>2</sup> See reference 739A for comparative pressure-volume curve of the two ventricles.

<sup>3</sup> Intracardiac phonocardiography in case of ASD reveals a systolic murmur in the outflow tract of the right ventricle but not in the atrium (1421).



### PROBABLE MECHANISM OF SHUNT IN A DEFECT

Fig. 336 The difference in the pressure-volume characteristics of the two ventricles is represented by balloons with different thicknesses of wall. Both are connected to the same pressure head by a T-tube. Obviously the thin-walled balloon dilates more.

of diastolic overload of the right ventricle exemplified by the condition under discussion atrial septal defect the characteristic electrocardiographic finding is incomplete right bundle branch block. Although usually the QRS does not attain or exceed 0.12 sec there is characteristically an R-R pattern in lead  $V_1$ . This finding does not occur in all cases of atrial septal defect as previously thought (ol 1137 p 293) but does occur in some two thirds of the cases. Milnor and Bertrand (110") found complete or incomplete bundle branch blocks in 17 of 24 proven cases of ASD. The incidence may be higher if statistics are based on  $V_m$ . Complete right bundle branch block occurs uncommonly.

In the ostium primum type of ASD there is often an associated mitral valve cleft with mitral regurgitation and strain on the left ventricle. In the electrocardiogram the combination of left axis deviation with incomplete right bundle branch block is rather characteristic of ASD of the ostium primum type with associated cleft of the mitral valve.

Pulmonary hypertension may develop in older patients with ASD because of wear and tear on the pulmonary vasculature (changes secondary to increased flow of long standing). At times pul-

monary hypertension may be present early, owing possibly to persistence of the fetal pattern of pulmonary vessels. Wood (1591) has what appears to be a different view of the pulmonary hypertension of ASD and many other conditions. He thinks that these individuals representing perhaps 20 per cent of all cases are persons whose pulmonary vasculature reacts in a vigorous and various manner to certain agents which do not ordinarily produce this effect unless present in extreme degree. It is suggested that the behavior is congenital. Wood found six cases of pulmonary hypertension (pulmonary pressure 43 to 81 mm Hg) among 50 cases of ASD. The duration of the hunt he thought could not be held responsible since the youngest subject was 21 years old and the average of the six was 16 years, less than that of the rest of the group. Welch and Kinney (124) in cases of ASD as well as patent ductus arteriosus and ventricular septal defect were unable to get evidence in support of the wear and tear effects of high flow.

The dilatation of the pulmonary artery is secondary to the increased pulmonary flow and is not necessarily dependent on the existence of pulmonary hypertension. There is usually a pressure gradient across the pulmonary valve. This

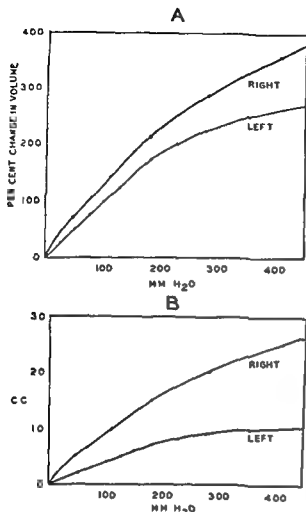


FIG 337 Pressure volume characteristics of atrial septal defect. *A* Relative curves. *B* Absolute curves. (From Little (1957))

is interpreted as representing relative pulmonary stenosis. There may be as much of a discrepancy as 35 mm Hg or even more (1061A) between systolic pressure in the right ventricle and pulmonary artery. The pressure loss in these cases is probably represented by laminar velocity, i.e., there is a conversion of pressure energy to kinetic energy. Normally, kinetic energy may represent as little as 5 per cent of the total made available by the contracting ventricle. In cases of ASD the figure may be several times this value. Indirectly, the high value for kinetic energy is related to the systolic murmur which is produced in the outflow tract and/or pulmonary artery. Therefore the pressure gradient across the pulmonary valve should bear some relationship to the intensity of the murmur.

CARDIOVASCULAR SOUND Atrial septal defect is

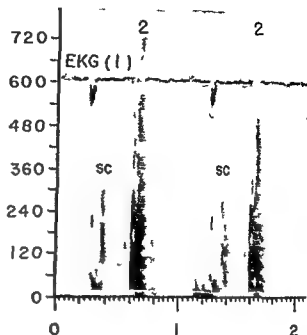


FIG 338 Pulmonary area with atrial septal defect. The first heart sound is virtually absent. The conspicuous early systolic click was confused for an unusually snappy S<sub>1</sub>. S<sub>1</sub> is split into two components (pulmonary closure) is greatly accentuated. The early systolic click initiates a soft systolic murmur. A faint early diastolic murmur follows the accentuated pulmonary closure sound.

to the group of congenital malformations where mitral stenosis is to be required valvular lesions. In all their ramifications the auscultatory phenomena illustrate principles which are pertinent in connection with most forms of congenital heart disease. The features requiring discussion are at least ten in number:

- 1 The tricuspid diastolic rumble
- 2 The pulmonary systolic murmur
- 3 The pulmonary early systolic click
- 4 The mid systolic click
- 5 The pulmonary early diastolic murmur
- 6 The split second heart sound
- 7 The split first heart sound
- 8 The tricuspid opening snap
- 9 The additional features of the ostium primum variety of ASD
- 10 The so-called phenomena which simulate those of mitral stenosis

A mid diastolic murmur may be present at the apex which is usually the right ventricle in these cases, or some point between the apex and the left sternal border. It is the auscultatory charac-

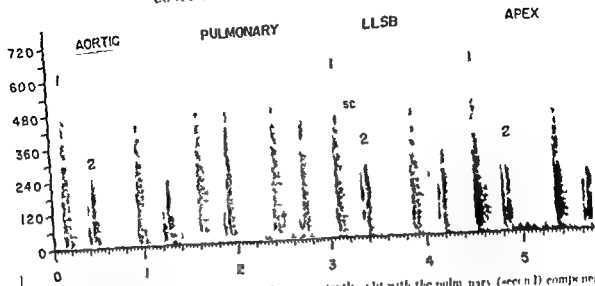


FIG 300 Atrial septal defects in V (60624) S<sub>1</sub> consistently split with the pulmonary (second) component very loud so that it is audible in both the aortic and the apical area. Mid systolic click at LLSB probably extra cardiac S<sub>2</sub> is unusually loud.

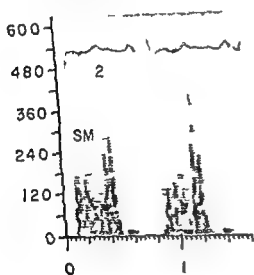


FIG 310 Split S<sub>2</sub> in ASD LLSB

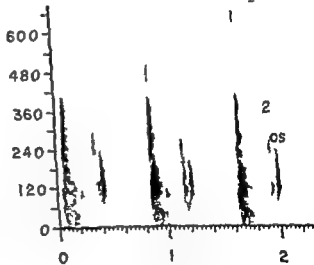


FIG 311 Mitral opening snap

For comparison with the split second sound shown in preceding figures S<sub>2</sub> followed by opening snap at apex in patient with mitral stenosis. Note ringing M<sub>1</sub> usually each component of a split S<sub>2</sub> has the appearance of a valvular closure sound with a high tone. For example Opening snap have a purer frequency content and usually the frequency bottom does not extend to zero.

teristics of a Carey-Coombs murmur (p 199). Barber, Magidson and Wood (51) found it to be audible in 9 of 62 cases of ASD—a third sound was present in 26. Blount and colleagues (120) described the disappearance of a mid diastolic murmur at the fourth left intercostal space after complete surgical closure of the defect. There is usually no pre-systolic accentuation. The murmur is explicable on the basis of the combination of high flow across the tricuspid valve (what Wood (1959) has termed 'torrential flow') and dilata-

tion of the right ventricle. Pre and colleagues (1234) call it a hypervolemic murmur. Nadasy (1137 p 280) states the rule of thumb that to get a diastolic rumble the pulmonary flow must be at least twice systemic flow. Presumably the

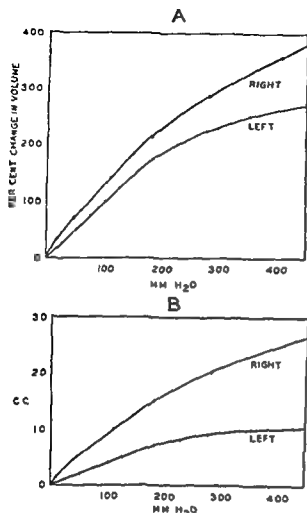


FIG. 337 Pressure-volume characteristics of atria. *A* Relative curves. *B* Absolute curves. (From Little (1957).)

is interpreted as representing, relative pulmonary stenosis. There may be as much of a discrepancy as 35 mm Hg or even more (106/1) between systolic pressure in the right ventricle and pulmonary artery. The pressure loss in these cases is probably represented by gain in velocity, i.e. there is a conversion of pressure energy to kinetic energy. Normally kinetic energy may represent as little as 5 per cent of the total made available by the contracting ventricle. In cases of ASD, the figure may be several times this value. Indirectly, the high value for kinetic energy is related to the systolic murmur which is produced in the outflow tract and/or pulmonary artery. Therefore the pressure gradient across the pulmonary valve should bear some relationship to the intensity of the murmur.

CARDIOVASCULAR SOUND Atrial septal defect is

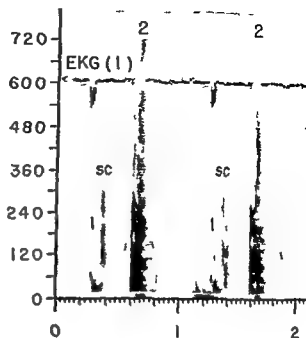


FIG. 338 Pulmonary area with atrial septal defect. The first heart sound is virtually absent. The conspicuous early systolic click was confused for an unusually snappy S<sub>1</sub>. S<sub>2</sub> is split, its second component (pulmonary closure) is greatly accentuated. The early systolic click initiates a soft systolic murmur. A faint early diastolic murmur follows the accentuated pulmonary closure sound.

to the group of congenital malformations what mitral stenosis is to acquired valvular lesions in all their ramifications the auscultatory phenomena illustrate principles which are pertinent in connection with most forms of congenital heart disease. The features requiring discussion are at least ten in number:

- 1 The tricuspid diastolic rumble,
- 2 The pulmonary systolic murmur,
- 3 The pulmonary early systolic click,
- 4 The mid-systolic click,
- 5 The pulmonary early diastolic murmur,
- 6 The split second heart sound,
- 7 The split first heart sound,
- 8 The tricuspid opening snap,
- 9 The additional features of the osium pium variety of ASD,
- 10 The some phenomena which simulate those of mitral stenosis.

A mid-diastolic murmur may be present at the apex which is usually the right ventricle in these cases or some point between the apex and the left sternal border. It has the oscillatory charac-

## CONGENITAL CARDIOVASCULAR DISEASE

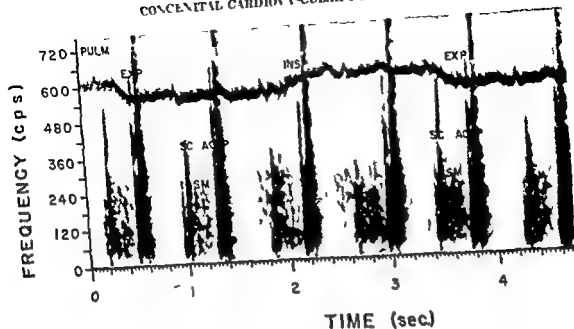
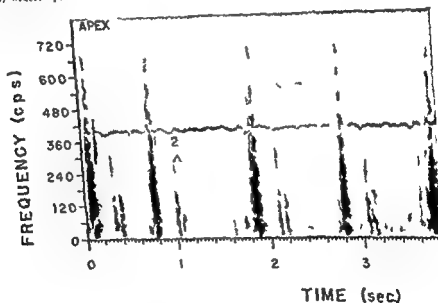


FIG 343 Atrial septal defect

Pulmonary area in D D (364143) 36 year-old female with long mitral regurgitation and aortic stenosis. Features typical of ASD: (1) early systolic click, maximal in expiration (2) decreased systolic murmur (3) widely split S<sub>2</sub>.

FIG 344 Variable S<sub>2</sub> splitting in ASD

P B (103156) 5 year old man was proved by catheterization to have ASD. Atrial fibrillation was present. After longer diastolic periods the degree of splitting of S<sub>2</sub> is greater.

listen to the same patient and hear only so unimpressive a systolic murmur that the competence of the previous observer is questioned. That the murmur indeed has origin in the pulmonary artery and not at the septal defect is supported by the

physiologic considerations discussed above. Furthermore, Rummel (1903, p. 396) found that in dog with surgically created ASD the microphone found the murmur over the pulmonary artery, not over the atrium and by intracardiac phono-

thinks this approximation holds for both the right side in ASD and anomalous venous return and the left side in VSD and PDA

It is possible that a mid diastolic murmur is sometimes generated at the septal defect. The remnants of septal tissue, which sometimes traverses the defect, and the lip of remaining septum

may be set into vibration. Iwasaki (1973) recorded mid diastolic murmurs over the base of the heart and attributed them to the mechanism mentioned

The pulmonary systolic murmur is the result of high flow through the outflow tract during ventricular ejection. Its intensity is highly variable from patient to patient and at various times in the same patient. For example, a patient may have a systolic thrill and systolic murmur at the left sternal border so loud that ventricular septal defect is considered. At another time one may

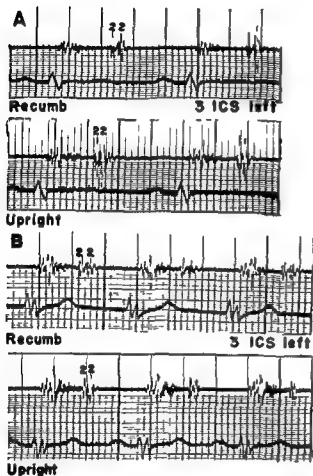


FIG 342A

FIG 342A 1 Decrease in splitting of  $P_2$  of ASD when subject upright. Phonocardiogram and lead II of the electrocardiogram of a 34 year old man with atrial septal defect. The calculated left to right shunt was 6.62 L per minute and the right ventricular pressure was 49/8 mm Hg. Note the wide splitting of the second sound (0.06 second) in the recumbent position and a decrease in splitting (0.03 second) in the upright position. The heart rate is almost identical in both tracings. B Phonocardiogram and lead II of the electrocardiogram of a 24 year old woman with atrial septal defect. The calculated left to right shunt was 8.96 L per minute and the right ventricular pressure 42/10 mm Hg. The electrocardiogram shows an incomplete right bundle branch block with QRS duration of 0.11 sec. Note the wide splitting of the second sound (0.07 sec) in the recumbent position and a decrease in splitting (0.04 sec) in the upright position. The heart rate is nearly identical in both tracings.

FIG 342B A Increase in  $S_2$  OS interval in MS when subject upright. Phonocardiogram and lead II of the electrocardiogram of a 42 year old woman with rheumatic heart disease and mitral stenosis. The calculated mitral valve area (Gorlin's formula) was 0.6 cm<sup>2</sup> and the pulmonary artery pressure 56/23 mm Hg. Note that the duration of the interval between the second sound and the opening snap (OS) measures 0.06 sec in the recumbent and 0.09 sec in the upright position. The heart rate is nearly the same on both occasions. B Phonocardiogram and lead II of the electrocardiogram of a 43 year old woman with rheumatic heart disease and mitral stenosis. Calculated mitral valve area = 1.2 cm<sup>2</sup>, pulmonary artery pressure = 37/23 mm Hg. Note that the duration of the interval between the second sound and the opening snap (OS) measures 0.09 sec in the recumbent and 0.11 sec in the upright position. The heart rate is more rapid in the upright position. Courtesy of Surawicz and Circulation (1451)

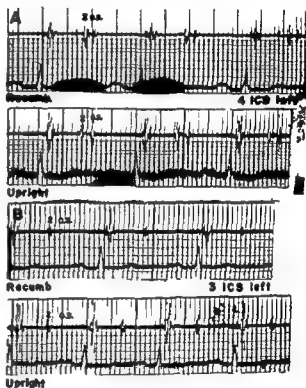


FIG 342B

enormously dilated pulmonary arteries coming into contact with the interior chest wall it vigorous pulsation producing the friction sound.

The split second heart sound is a highly characteristic feature of ASD. It was audible as grade III splitting in 52 of 62 cases (84). It has been

in the discrepant stroke volumes—and therefore systolic duration—of the two ventricles. Pulmonary valve closure is delayed in relation to aortic valve closure. It has long been appreciated that increase in venous return and increase in stroke volume prolongs mechanical systole of the ventricle (pp 117 to 122). The split  $S_2$  of ASD is a peculiar striking persistent and pathologic variant of a phenomenon seen in most normal individual—namely splitting of the second sound with inspiration owing to the increase in venous return.

Since it is now clear earlier occurrence of aortic closure is responsible for part of the normal inspiratory splitting of  $S_2$  (p 119) the absence of change in the timing of aortic closure in cases of large ASD with fixed splitting of  $S_2$  requires explanation. It is entirely plausible to presume that because of the congestion of the pulmonary circuit and the fact that the two atria are functionally one in inspiration will not result in the usual reduction in left ventricular filling in ASD. In some cases although the interval between the aortic and pulmonary components is fixed both components fall slightly later in inspiration than in expiration (147). The explanation afforded is that the venous return from the lung is not reduced in these cases or if anything is increased (because of the pulmonary hypertension) that the

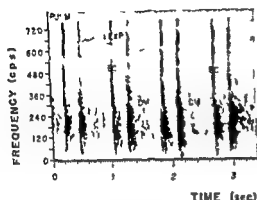


FIG. 30 ASD with pulmonary regurgitation.

Pulmonary area in F.W. (19311) 13 year old female with ASD and pulmonary hypertension. Record made 18 months after closure of the septal defect. The loud early systolic sound is too late for  $S_1$  and must be early systolic click  $S_1$  split with its second component tremendously accentuated and followed by a decrescendo murmur of pulmonary regurgitation. Cardiac catheterization showed no change in pulmonary pressure (about 80/20 mm Hg) after the operation.

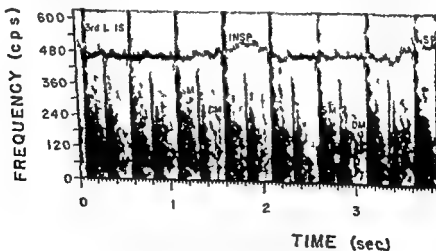


FIG. 31 ASD with pulmonary regurgitation.

This left interspace in F.W. (B35) 13 year old female  $S_1$  loud there is a decrescendo ejection systolic murmur  $S_1$  characteristically split and the second (pulmonary) component is followed by a decrescendo systolic murmur. EKG was that of right ventricular hypertrophy not right bundle branch block in this case of ASD with pulmonary hypertension.



cardiography in patients with ASD Soulié *et al* (1421) found the murmur in the pulmonary artery, not in the atria

Transmission of the systolic murmur to the inter-scapular area of the back is a diagnostically helpful feature. Usually the systolic murmur of ASD is about as loud in the back as anteriorly. Corbitt is one of the few other conditions which show this phenomenon. The murmur of pulmonary stenosis when very loud is as one would predict, well heard in the back. However, confusion is not likely to be created thereby.

The early systolic click (Fig 338) represents snapping of the pulmonary arterial wall early in ejection. In no other single condition does it occur more consistently than in ASD. By phonocardiograms it can be demonstrated in virtually all cases. Its characteristics have been described in detail elsewhere (see p 179).

In one study a mid systolic click was recorded from the left sternal border in many cases. It was attributed (1082) to movement of the costochondral and/or chondrosternal joints by the hypertrophied right ventricle, i.e. it was thought to be related to the left parasternal heave. Since the finding has not been noted on stethoscopy and other phonocardiographic studies have not com-

mented on it, there is a possibility that the mid systolic click is an artifact characteristic of the microphone or of the recording and analyzing set up used in this study, when exposed to the intense impulse of the hypertrophic right ventricle.

A diminuendo pulmonary diastolic murmur (Figs 346 and 347) is heard occasionally. In one series it was audible in 36 of 62 cases (58) but in most series the incidence has been less. Bedford and colleagues (80) found it in 20 per cent. I found it in 2 of 11 cases (1082), in one the murmur was accentuated in inspiration. It has its basis in dilatation of the pulmonary ring as part of the general enlargement of the pulmonary artery. It may persist after closure of the atrial septal defect, particularly in adults and in children with pulmonary hypertension.

Coulshed and Littler (306) in describing five instances of ASD in aged patients (58 to 79 years) reported that in one an alleged pericardial friction rub persisted for five years! It had a superficial quality and became louder with pressure on the stethoscope. Although it may be that systolic and diastolic murmurs were incorrectly interpreted as a pericardial friction rub the authors suggested that it may have been produced by the

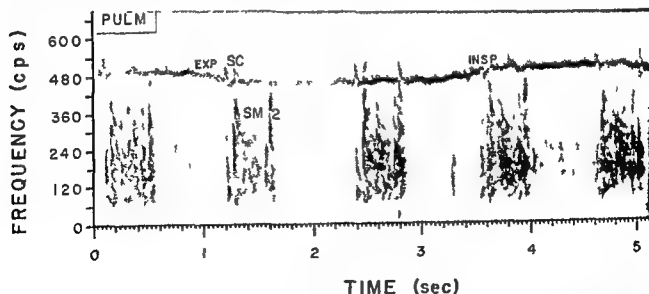


FIG 345 ASD with normal I EKG

Pulmonary area in J H B (75084) 27 years old with ASD (or AVR) proven by cardiac catheterization but with normal EKG. The findings are typical of ASD (or possibly AVR) (1) early systolic click followed by (2) a decrescendo systolic murmur and (3) a widely split  $S_2$  of which the second (pulmonary) component is louder. Splitting of  $S_2$  is exaggerated in inspiration. The findings indicate that incomplete right bundle branch block is not invariable in ASD and that there may be respiratory variation in the splitting of  $S_2$  in cases of mild ASD.

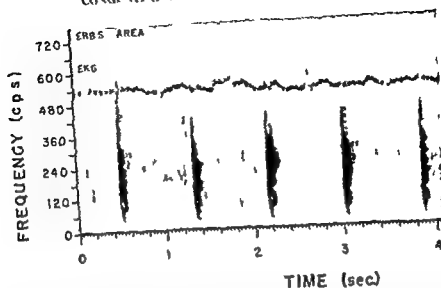


FIG. 319 (91) postoperative possible Lutembacher syndrome

R F S L (68/49, 68/25) 30-year-old had closure of an ASD 7½ years previously. Catheterization failed to demonstrate any evidence of persistent hunt. The SIC (Irb) area demonstrated per cent of lifting of  $S_1$  which is however of minor degree and shows normal exaggeration with inspiration. There is an early diastolic murmur with the characteristics of an AV opening snap. Furthermore, it is probably of mitral origin since it maintains a constant relation ship to the first (aortic) component of  $S_2$ . The mitral valve may be involved in this patient—Lutembacher's syndrome.

because the tricuspid valves are widely separated by the torrential tricuspid flow which is likely to continue through the entirety of diastole. The wider separation of the leaflets accounts not only for the delay in the sound of closure but also for the accentuated and ringing character which this sound frequently has. The combination of ringing tricuspid closure sound following a normal or somewhat reduced mitral closure sound gives a cardiac contraction to the first sound (Fig. 348). This combination may simulate the presystolic murmur and ringing first sound of mitral stenosis.

Leatham and Gray (863) found a snap with the proper temporal relationships for an AV opening snap presumably a tricuspid opening snap. There are other instances (e.g. patent ductus and ventricular septal defect) to indicate that not only can a diastolic murmur of relative stenosis be attributed to torrential flow, but also an opening snap. It is quite credible that even a normal valve might produce a nappy sound if opened vigorously.

One of the clinical clues to the presence of the ostium primum variety of ASD is the presence of

the systolic murmur of mitral regurgitation with radiation to the left axilla. However, the AV values may be normal as have been observed in two cases (ZO 673149, B2 (96212) in which the auscultatory findings were no different from those of the secundum variety of ASD (see Fig. 131).

Lutembacher's syndrome—mitral stenosis (acquired or congenital) and ASD—is uncommon. An exaggerated impression of its incidence is created by a galaxy of auscultatory and other clinical features which suggest mitral stenosis in cases of pure ASD. Quite aside from the fact that the radiologic configuration of the heart (except for the absence of left atrial enlargement) and the EKG findings are often consistent with mitral stenosis, there are the following some features mimicking mitral stenosis:

- 1 Crescendo first sound with accentuated tricuspid element suggesting presystolic murmur and ringing mitral first sound (see above).
- 2 Split second sound suggesting second sound proper plus mitral opening snap.
- 3 Mid-diastolic rumble—although of tricuspid origin—is often in the region of the apex (which in this condition is likely to be the right ventricle).

two ventricles are filled from a common reservoir, and that the effective filling pressure of both ventricles is increased in the manner that only the right ventricle is influenced normally during inspiration.

In most cases of ASD (except those with relatively small shunts) the degree of splitting is constant ("fixed") is becoming the cliché and displays no variation with respiration (Fig. 344). There may be some reduction in the degree of splitting when the subject is in the standing position as compared with the recumbent position (1454). Marked variation in the degree of splitting with inspiration and posture suggests that the diagnosis is not ASD, or that the shunt, if present, is small. The splitting of  $S_2$  in ASD is not dependent on the presence of bundle branch block (Fig. 345) complete or incomplete (7) though it may be increased with complete right branch block), or on the presence of pulmonary hypertension (although the splitting tends to be reduced in such cases). In fact with pulmonary hypertension the splitting may be less because the volume of the left to right shunt is reduced (1591).

Seemingly no one has attempted a systematic correlation of the volume of the shunt (or of the discrepancy between pulmonary and systemic flow) with the degree of splitting. Splitting of  $S_2$  was not found by Bertrand et al. (96) in dogs with experimentally produced ASD. Rogers

et al. (1307) found it some weeks after creation of the defect.

Blount and colleagues (120) observed disappearance of fixed splitting after surgical closure of the defect. Wood (1591A) and many others have made similar observations. Wood states the splitting may still be present when the subject is recumbent but not when he is upright. He thinks reduced resistance to right ventricular filling may persist for a time after operation.

Intensity of  $S_1$  is not a reliable index of the level of pulmonary pressure since, as in other disorders leading to dilatation of the pulmonary artery, intensification appears to be related to proximity of the vessel to the anterior chest wall.  $P_2$  is commonly loud in ASD, regardless of the level of pulmonary artery pressure. Wide separation of the cusps just before closure, as a result of the high flow, may be another factor in the loud  $P_2$  of ASD.

The first heart sound is also split at times (Fig. 154), although this is a less consistent feature than splitting of the second sound. This splitting is the result of a delay in the tricuspid closure sound. Normally, mitral closure slightly precedes tricuspid closure. In ASD this is exaggerated. The mechanical lag between onset of right ventricular contraction and tricuspid valve closure is increased perhaps in part because of slower rise in ventricular pressure but probably mainly

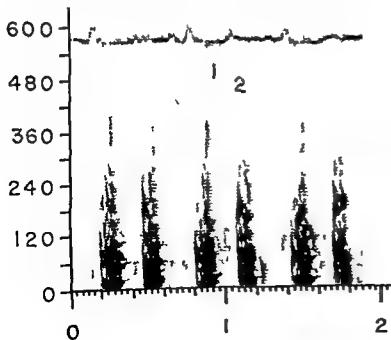
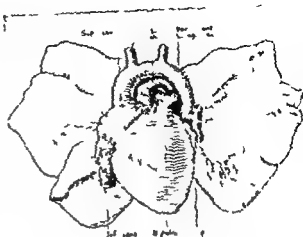


FIG. 345. Centric construction of  $S_1$  with ASD probably caused by the first sound followed by an early aortic click of pulmonary origin. In other cases it may be caused by a mitral closure sound followed by an accentuated tricuspid closure sound. Whatever the basis mitral stenosis may be simulated. (Note plot S. H. S. in D. A. (34202))

FIG. 351 Total anomalous pulmonary venous return. Sketch of an anatomic specimen in a patient with figure of eight syndrome. All venous drainage from the right lung is into a transverse venous mass which courses posterior to the heart and joins the right left superior vena cava that receives the venous drainage from the left lung. Obviously all pulmonary venous return is to the right side of the heart and ASD is necessary present. (Reproduced from MacMahon (101) with the permission of the author and publisher.)



which appears to be holosystolic because it extends up to the aortic closure sound. Often the systolic murmur of mitral regurgitation is also heard in the axilla (1140).

#### ANOMALOUS PULMONARY VEIN RETURN (AVR)

(558) Anomalous pulmonary venous drainage or connection in position of the pulmonary vein.

**DEFINITION.** Included in this category is a number of malformations in which at least part of the venous return from the lungs enters the right side of the heart rather than the left (1076).

**ANATOMICAL CONSIDERATIONS.** There are two main categories of anomalous venous return: (1) partial (2) total. In the latter group the presence of an atrial septal defect is essential to survival. The varieties of anomalous venous return are illustrated by the classification of cases seen in this hospital and presented in Table 14 (p. 122). The figure of eight variety is illustrated by the sketch in Figure 351 and the trans studies in Figures 352, 357 and 358.

**PHYSIOLOGICAL CONSIDERATIONS.** Physiologically and clinically AVR particularly the partial type has many features identical to those of ASD. As ASD there is a left to right shunt at the level of the atrium. At cardiac catheterization it is often impossible to differentiate the two disorders in both an oxygen step up may be discovered in the right atrium and the catheter may pass into the lung field from the area of the right atrium.

Cardiovascular studies. Because of the physiologic parallels to ASD all the same changes in the heart sound are to be expected. For example a mid-diastolic murmur at the apex (52) (238) (173). As in ASD because of the left to right shunt pulmonary blood flow may be much greater than systemic. It is little wonder then that there may be a striking pulmonary systolic murmur.

Occasionally in total AVR of the transverse figure of eight or catgut knot variety (so called because of the radiologic appearance created by the left sided superior vena cava and the dilated though normally positioned right superior vena cava) a continuous murmur (phonocopyically and phonocardiographically identical to that of patent ductus arteriosus) may be heard under the right clavicle (777). Anomalous right sided PDA has been mistaken diagnosis in certain of these cases (see Fig. 159). The site of generation of the murmur is clearly the point where the persistent left superior vena cava enters the normal superior vena cava as demonstrated in Figure 356 and 357 angiograms show striking dilation at this site. The superior vena cava usually has a bulge to the right at this site and pulsates very noticeably up against an arterial structure. In fact in roentgen kymograms or electrokymograms the character of the pulsations may appear to be arterial. Further more at operation exposure of the vena cava reveals a bulge over the superior vena cava and

Actually the left superior vena cava empties into the anomalous vein which in turn joins the right superior vena cava.

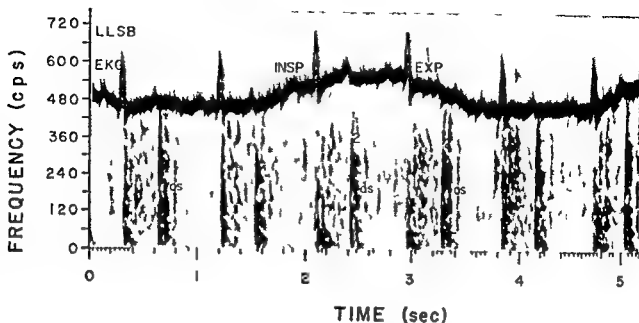


FIG. 350 Opening snap in atrial septal defect

A. B. (792815) a 38 year old woman had a story of indubitable rheumatic fever. The clinical signs except for the opening snap were characteristic of atrial septal defect. As were all the findings of cardiac catheterization. The patient died five days following open heart surgery for closure of the atrial septal defect. Necropsy revealed very minimal fibrosis in the aortic leaflet of the mitral valve and associated chordae tendineae.

The basis of the opening snap was not clear in this case. The mitral valve could not be completely exonerated. It should be noticed that the first sound at the apex was normal in quality and in timing.

Nadas and Alimurung (1138) found an apical diastolic murmur in 19 of 100 patients with simple ASD. Among 20 patients with congenital malformations of the heart and apical diastolic murmurs the diagnosis was ASD in 8, VSD in 5, PDA in 4, Eisenmenger complex in 2 and single ventricle in 1.

4. Early systolic click which also occurs commonly with the dilated pulmonary artery of mitral stenosis.

5. The occasional occurrence of an AV opening snap of probable tricuspid origin in ASD increases the simulation of mitral stenosis.

#### ENDOCARDIAL CUSHION DEFECTS

(Common atrioventricular canal, AV communis, persistent common AV canal, ostium primum)

**ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS**  
The designation suggested for this general group by Campbell and Milten (228) is used. All members of the group result from faulty development and fusion of the dorsal and ventral AV endocardial cushions. The result is at one extreme a persistent common AV canal in which there is a

single anterior and a single posterior AV cup, each common to the two ventricles, and a single AV orifice continuous with a persistent ostium primum above and a high ventricular septal defect below. The other extreme result is persistent ostium primum. Although ostium primum defect with normal AV valves is reported (cf. case 3 of Blount (118)), the usual experience is an associated cleft of the anterior aortic leaflet of the mitral valve (1499). Occasionally the tricuspid valve is also affected. Discussion of this group is introduced at this point because clinically atrial septal defect is simulated. The shunt is usually from left to right and the patients are asymptomatic. Defects of this type occur commonly in association with mongoloid idiocy.

**CARDIOVASCULAR SOUND** The fixed splitting of S seen with secundum type of ASD occurs also with the primum type. A systolic murmur of mitral regurgitation is common. When the other features of ASD are present, a loud systolic murmur which is well heard in the left axilla is a valuable clue to identification of the defect as an ostium primum variety. One must not be confused by a pulmonary ejection systolic murmur

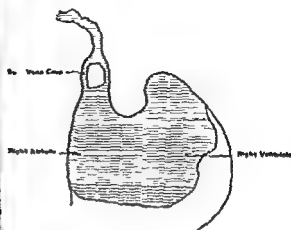


FIG 331 Anomalous pulmonary venous return

Film from an angiographic series illustrating dilution of the inferior vena cava at the point where the anomalous pulmonary vein enters it. A murmur is likely to be generated at this point.

great swirls are visible where the fully oxygenated blood returning from the lung meets the dark venous blood (e.g. K F 30400).

The essential feature of the superior venous murmur in total AVR, the feature which results in simulation of IDA, is its maximum in late systole. It might be the flow meter observation of Brocher (172) and of Nilsson and Kræmer (114) that flow in the superior vena cava is maximal during ventricular systole, presumably because the atrioventricular diaphragm is pulled down markedly during this phase.

Although in some cases the murmur is loudest on the left of the sternum (in these instances it probably arises in the left SVC) and has the flickiness and the humming quality of a venous hum, the murmur mentioned above simulates that of PDA precisely except for its location.

The murmur of pulmonary regurgitation is described in some cases (192) (1101). I have been surprised by the infrequency of fixed splitting of S<sub>2</sub> in cases of AVR. Ongley (1139) makes the same statement. Many other authors do not mention splitting of S<sub>2</sub> as a feature of these cases. Occasionally writers (e.g. 336) have mentioned it

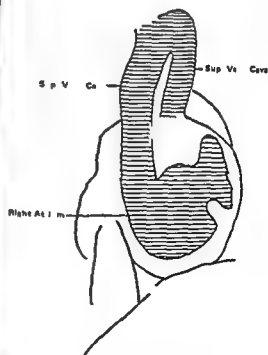
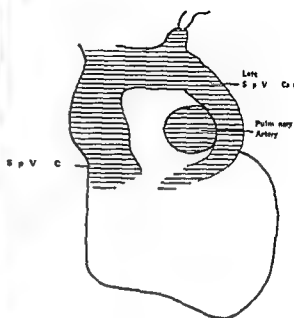
## VENTRICULAR SEPTAL DEFECT

including the Eisenmenger complex and the syndrome of aortic regurgitation.

(See Roger's disease, *maladie de Roger VSD*.)

**DEFINITION.** Any communication between the two ventricles via one or more foramina in the ventricular septum.

**ANATOMIC CONSIDERATIONS (77, 1309).** The anatomical variability is the unappealing feature. More involved primarily the membranous septum than the muscular septum. However, even in the instance of involvement of the membranous septum there is likely to be some involvement of the muscular septum as well. There is little basis for the old concept that benign VSD (Roger's disease) is usually muscular or low, whereas serious VSD was high or membranous. The most frequent variety of defect appears to be one in which the membranous septum is primarily involved, the defect lies as far as the left side is concerned below the aortic valve in the portion of the septum that contributes to the outflow tract of



FIGS. 352(A) and 353(B) Total AVR

PA (352) and lateral (353) views made simultaneously in an angiographic series in a child with the figure of eight syndrome. Pulmonary veins demonstrated and superior loop opacified

TABLE 11

Types of anomalous venous connection (1076)

| Type   | No. |
|--|-----|
| Isolated transposition   |     |
| To inferior vena cava  | 1   |
| To superior vena cava  |     |
| Right superior pulmonary vein  | 3   |
| Left superior pulmonary vein   | 1   |
| To right atrium directly   | 1   |
| Left pulmonary vein(s) to right atrium via pericardial superior vena cava on left & coronary sinus | -   |
| Total transpositions   |     |
| To right atrium (unspecific left ventricle)  | 2   |
| To right atrium (via coronary sinus)   | 1   |
| Figure of eight valve (pericardial superior vena cava on left to superior vena cava on right)      | 1   |
| To superior vena cava on right via azyg vein   | 1   |

above the cri-ta supraventricular in the outflow tract of the right ventricle. Defect in the muscular septum are likely to be multiple.

Anatomically the Lecommer complex is the tetralogy of Fallot minus pulmonary stenosis. It differs from simple VSD in that there is dextro-position or overriding of the aorta. Physiologically the Lecommer complex has parallels to TETRALOGY OF FALLOT in that there is obstruction to pulmonary blood flow but the obstruction is located at the level of the pulmonary arterioles.

Acquired VSD occurs with myocardial infarction (see p. 123) and after stab wounds of the heart (211B, 477A, 1022A). Non-penetrating trauma to the chest is occasionally the cause of perforation of the septum (1420).

**PHYSIOLOGIC CONSIDERATIONS** Because of the greater pressure in the left ventricle than in the right the shunt in VSD is from left to right under ordinary circumstances. As a result pulmonary blood flow and mitral orifice flow (but not aortic flow) are likely to be greatly increased. Only during systole the pressure in the two ventricles is different with the higher pressure in the left ventricle. Therefore the shunt and murmur produced thereby occur only in systole.

The clinical manifestation and gravity of VSD are determined by the size of the defect and the resistances in the systemic and pulmonary cir-



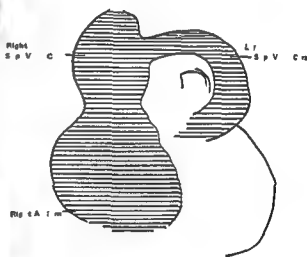
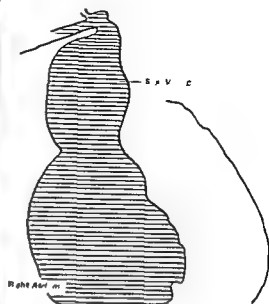
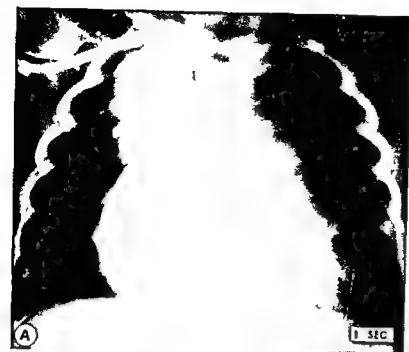
FIG. 3a (above) and 3b (below) Total A-V-R

Ordinary radiograms of the chest in patients with the typical figure of eight syndrome. The left part of the SVC (arrows in Fig. 3a) is always the least dense component of the anomalous cardiovascular stripe.

paragraph the intimate anatomical relationship to the aortic valve is evident. It is not surprising that retroversion of the right anterior coronary cusp with aortic regurgitation may occur from lack of proper suspension and support for that cusp.

Occasionally the communication is located





FIGS. 355(A) and 356(B) Total AVR

Two films from an angiographic series in K. I. (506600) a 7 year old child with continuous IIA like murmur under the right clavicle. Fig. 355 demonstrates the massive dilatation of the superior vena cava and right atrium. At the time that the pulmonary veins are demonstrated by contrast substance (Fig. 356) the left SVC is opacified and the right SVC and right atrium are re opacified. In the right SVC swirling is demonstrated in Fig. 356. At operation the swirling mixture of arterial and venous blood was striking.

the left ventricle, and as far as the right side is concerned, the defect communicates with the right ventricle under and behind the septal leaflet of the tricuspid valve. The stream of blood in such instances must pass among the tricuspid chordae tendineae. When bacterial endocarditis occurs in association with VSD the vegetations

are often located primarily on these chordae. There are often anomalous tendons traversing the right side of the defect with attachment at the upper and lower margins like the strings of an Aeolian harp (see Fig. 186 also, Gould (50) and Taussig and Semans (1462)).

In the variety of VSD described in the 1st



FIGS 3a (above) and 3b (below) Total A/R  
Ordinary radiograms of the chest in patients with the typical figure of eight syndrome. The left part of the A/C (arrows in Fig 3a) is always the least dependent of the anomalous cardiovascular tripe

paragraph the intimate anatomical relationship to the aortic valve is evident. It is not surprising that retroversion of the right anterior coronary cusp with aortic regurgitation may occur from lack of proper suspension and support for that cusp.

Occasionally the communication is located

TABLE II

Types of anomalous venous return (106)

| Type  | N. of Cases |
|---|-------------|
| Partial transposition                                     |             |
| To inferior vena cava                                     | 1           |
| To superior vena cava                                     |             |
| Right upper pulmonary vein                                | 3           |
| Left upper pulmonary vein                                 | 1           |
| To right atrium directly                                  | 1           |
| Left pulmonary vein ( ) to right atrium via               |             |
| pericardial septum  | 1           |
| coronary sinus  |             |
| Total transposition                                       |             |
| To right atrium (unspecific point of entry)               | 2           |
| To right atrium (via coronary sinus)                      | 1           |
| Figure of eight syndrome (pericardial septum)             | 8           |
| superior vena cava on left to superior vena cava on right |             |
| superior vena cava on right via azygos vein               | 1           |

above the crista supraventricularis in the outflow tract of the right ventricle. Defects in the muscular septum are likely to be multiple.

Anatomically the Eisenmenger complex is the tetralogy of Fallot minus pulmonary stenosis. It differs from simple VSD in that there is dextroposition or overriding of the aorta. Physiologically the Eisenmenger complex has parallel to TIA. In TIA there is obstruction to pulmonary blood flow but the obstruction is located at the level of the pulmonary arterioles.

Acquired VSD occurs with myocardial infarction (see p. 42) and after stab wound of the heart (21B, 177A, 1022A). Non-penetrating trauma to the chest is occasionally the cause of perforation of the septum (1120).

PHYSIOLOGICAL CONSIDERATIONS Because of the greater pressure in the left ventricle than in the right the hunt in VSD is from left to right under ordinary circumstances. As a result pulmonary blood flow and mitral orifice flow (but not aortic flow) are likely to be greatly increased. Only during systole is the pressure in the two ventricles different with the higher pressure in the left ventricle. Therefore the hunt and murmur produced thereby occur only in systole.

The clinical manifestations and gravity of VSD are determined by the size of the defect and the resistances in the systemic and pulmonary cir-

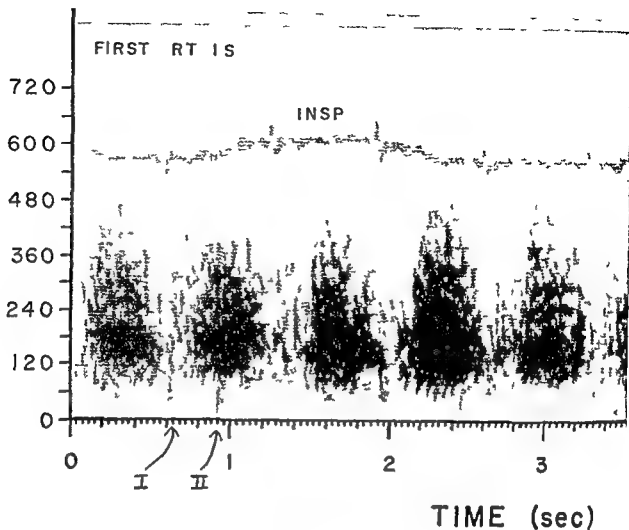


FIG. 359 Total anomalous venous return of figure of eight type. Intense murmur has greatest amplitude and frequency span in the region of the second heart sound (II) as in the murmur of patent ductus arteriosus. Recorded under midsaxial in first right inter space.

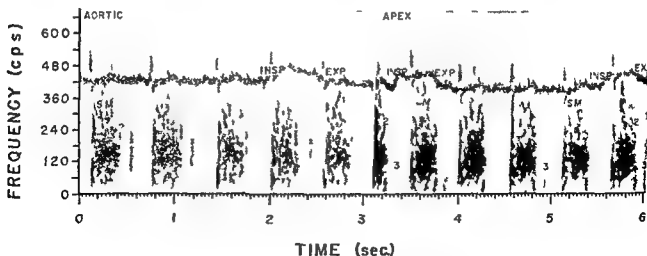


FIG. 360 Maladie de Roger

L. M. (759824) 5½ year old female demonstrated typical cardiac catheterization findings with a pulmonary artery pressure of 41/8 mm Hg. In the aortic apex as elsewhere there is a holosystolic murmur stopping with the first (aortic) component of S<sub>2</sub>. S<sub>2</sub> is minutely split in inspiration. A third heart sound gallop is demonstrated at the apex and is probably produced by hyperdynamic filling of the left ventricle. The snappy sound marked 'x' seems to occur later than the third heart sound and its nature is unclear. See Figure 362 for a similar finding.

## CONGENITAL CARDIOVASCULAR DISEASE

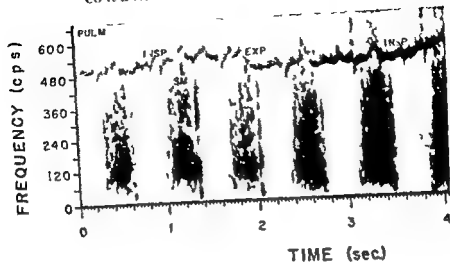


FIG 31 Mitral regurgitation

Pulmonary area in M L (Vib 4) 17 year old asymptomatic female. There was no mid-diastolic rumble at the apex. There is a loud holosystolic murmur at the left sternal border. In the pulmonary area  $S_1$  is slightly split with moderate accentuation of the second component which is probably pulmonary.

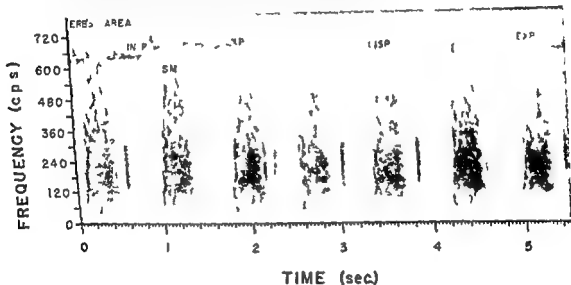


FIG 32 Ventricular septal defect

Third left inter-space in T R (Vib 9) 14 year old female. The murmur was discovered by the father (as a thrill) when he was holding the infant at the age of two weeks. FkC shows balanced axis and partial right bundle branch block. The heart is at the upper limit of normal for size with increased prominence of the pulmonary artery which has increased pulsation. The murmur extends throughout systole  $S_1$  is very lightly split. There is an early diastolic snap mainly in expiration which is not understood. See figs 140, 149 and 360 for diastolic clicks which like this may be extracardiac in origin.

On these factors depend the size and direction of the shunt and the level of pulmonary pressure (1376). Overriding of the aorta is probably only a minor determinant.

Whether with small defects—especially of the

muscular septum—closure of the defect with systolic contraction of the ventricle occurs can only be speculated. Factors which alone or in various combinations may reduce (or almost totally reverse) the right-to-left shunt include

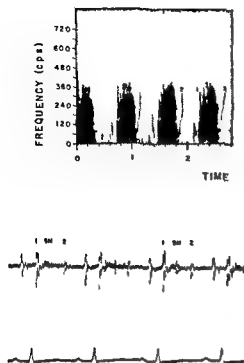


FIG 363 Ventricular septal defect

1601c Left sternal border in J T (276556) with VSD  
Musical element to murmur Pre systolic gallop (1)  
Below: Oscillographic PCG corresponding to 1

(1) increased resistance to pulmonary flow through narrowing of the pulmonary arterioles or from pulmonary stenosis and (2) dextroposition (overriding) of the aorta. As in the case of ASD, it is thought that two factors, alone or in combination, may be operative in producing the increased pulmonary vascular resistance: congenital anomaly, i.e., persistence of the fetal pattern of the small pulmonary vessels and changes from the wear and tear of increased flow.

Returning to the left ventricle through the mitral valve is the usual amount of blood swelled by the addition of that volume which is shunted through the defect and recirculated through the lungs. The mitral diastolic murmur is produced by this increased volume of blood. The dilated left ventricle contributes. The stroke volume of the left ventricle is increased and the diastolic volume by necessity increased.

In the Eisenmenger complex cyanosis usually *cyanosis tardus* often appearing first in the teens is present because of pulmonary hypertension. There are certain physiologic similarities to tetralogy of Fallot: the obstruction to pulmonary flow

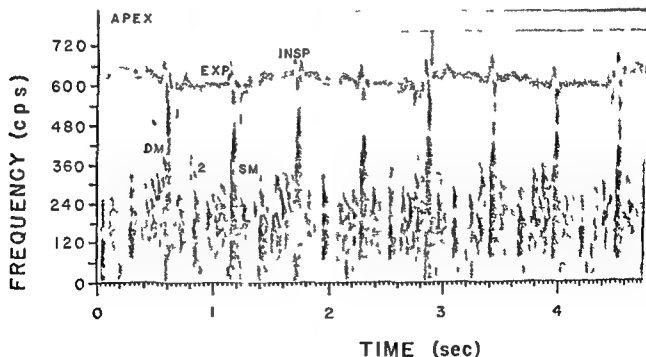


FIG 364 Carey Coombs murmur with VSD

I (B12914) 5½ year old female had sieve like interventricular septum with five perforations. There is an impressive diastolic murmur beginning after a gap following S₂ and displaying a pre-systolic crescendo. The mitral valve was normal.

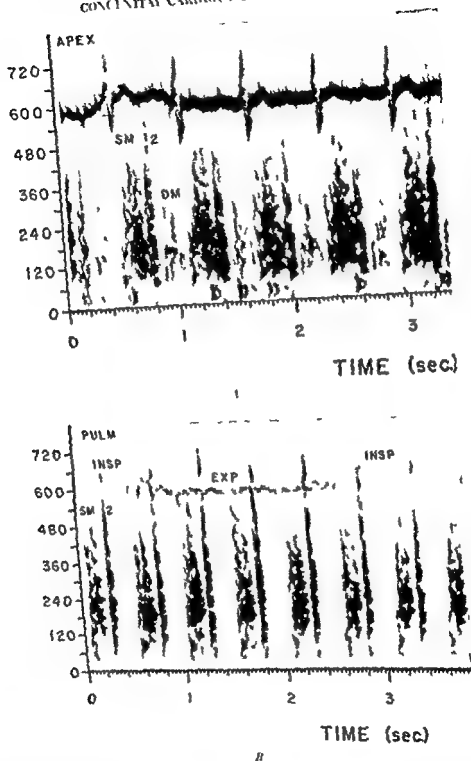


FIG. 363 ASD with pulmonary hypertension

SM II (Bo 09) had SM II at the age of 4 years. At the age of 5 years the diagnosis of ASD (with systolic pulmonary pressure of 80 mm Hg) was established by cardiac catheterization. There is a loud mid diastolic rumble at the apex. The systolic murmur in the pulmonary area is not quite holosystolic as it usually is with this degree of pulmonary hypertension.

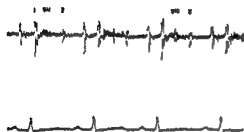
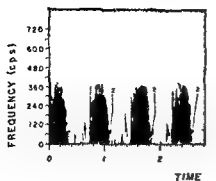


FIG 363 Ventricular septal defect

Above Left sternal border in J T (2765/6) with VSD  
Musical element to murmur Presystolic gallop (A)  
Below: Oscillographic PCG corresponding to 1

(1) increased resistance to pulmonary flow through narrowing of the pulmonary arterioles or from pulmonary stenosis and (2) dextroposition (overriding) of the aorta. As in the case of ASD, it is thought that two factors, alone or in combination, may be operative in producing the increased pulmonary vascular resistance: congenital anomaly, i.e., persistence of the fetal pattern of the small pulmonary vessels and changes from the wear and tear of increased flow.

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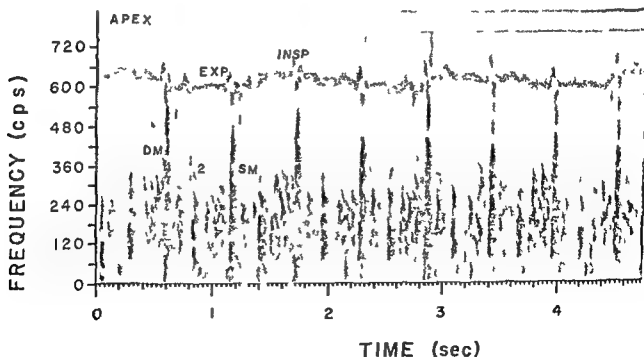


FIG 364 Carey Coombs murmur with VSD

J C (B12914) 5½ year old female had sieve like interventricular septum with five perforations. There is an impressive diastolic murmur beginning after a gap following S<sub>2</sub> in 1 di playing a pre-systolic crescendo. The mitral valve was normal.

in the pulmonary arterial tree rather than pulmonary valvular area.

Corlin formula for estimating orifice size has been used to estimate the size of ventricular defect (1952).

Some maintain that pulmonary resistance tend to increase progressively in VSD with large shunt. On the other hand Wood and his colleagues insist that high pulmonary resistance does not develop slowly over the years but is determined at or shortly after birth. As cardiac catheterization ages in answer to this difference of opinion will be forthcoming. Truth may be found in both statements. A fascinating recent observation (227, 228) is that of acquired pulmonary stenosis in VSD a phenomenon which is demonstrated by

repeat catheterization and accounts for clinical improvement after the first year.

**CARDIOVASCULAR SOUND** The typical Roger murmur is holosystolic intense burying both the first and the second sound maximally at the left sternal border at about the level of the third and fourth interspaces. The murmur is characteristically accompanied by a thrill in the same area. There may be a musical element to the murmur discernible by ear and SPCG against the noisy background. At the apex there may be a Carey Coombs type of blubbery, mid-diastolic murmur due to torrential mitral flow.

When there is increased resistance to pulmonary flow the systolic murmur tends to become shorter i.e. limited to the first part of systole and may disappear completely. As pulmonary resistance increases the pattern of contraction in the right ventricle comes to resemble that in the pure pulmonary stenosis. Peak pressure is attained late in systole. Consequently if the pattern of left ventricular systole remains unchanged, pressures are gradient across the septal defect will exist only in the first part of systole.

There is reason to think that there is often a pulmonary early systolic click and a separate systolic murmur due to dilated pulmonary arteries.

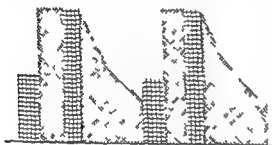


FIG. 68 VSD with aortic or pulmonary regurgitation

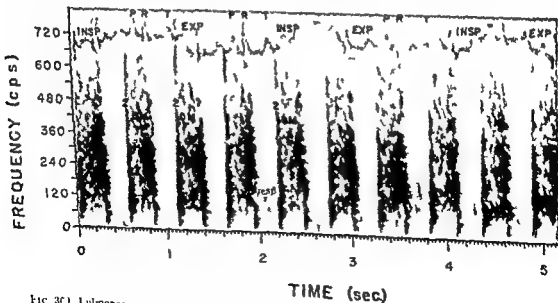


FIG. 3C) Pulmonary regurgitation in VSD with severe pulmonary hypertension and reverse shunt. 11 SB in SC M (252896 46 413) 30 year old female has hemophilia as well as evanescent congenital heart disease. Repeatedly, none of her have misinterpreted her murmur as systolic. Her rate is always rapid with a lot of late diastolic and early diastolic and the murmur is not typically decrescendo in fact it becomes louder in



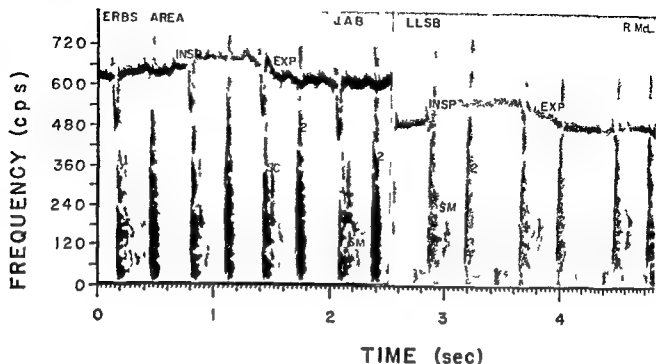


FIG 366 VSD with pulmonary hypertension

J A B (750002) 10 year old female had the tentative diagnosis of ASD. Cardiac catheterization showed left right shunt at the ventricular level and marked pulmonary hypertension (122/73 mm Hg). The absence of splitting of  $S_2$  would tend to exclude ASD without catheterization. The early systolic click and absence of systolic murmur are consistent with VSD with pulmonary hypertension or with pulmonary hypertension alone. LLSB in R MCL (75003) 25 year old male with VSD proved by cardiac catheterization which also showed the pulmonary artery pressure to be 140/68 mm Hg. The recordings in other areas were essentially the same as shown here. Of note are (1) the accentuated (2) short uncompressive early systolic murmur suggesting that of high pulmonary artery flow and/or pulmonary artery dilatation.

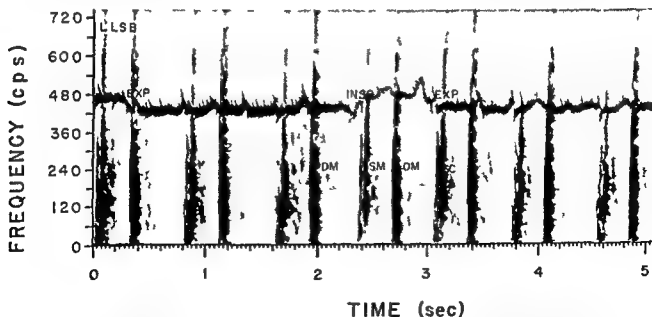


FIG 367 VSD with pulmonary hypertension and pulmonary regurgitation

D M S (760118) 13 year old female had findings of cardiac catheterization consistent with VSD. pulmonary artery pressure was 100/48 mm Hg. In this recording, from the left sternal border note the loud  $S_2$  followed by decrescendo early diastolic murmur and the early systolic click followed by a murmur which has the character of an ejection systolic murmur and may in fact be produced by the right ventricular outflow rather than flow through the VSD. Systemic diastolic pressure was normal.

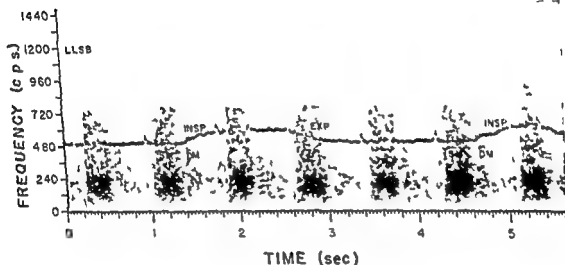
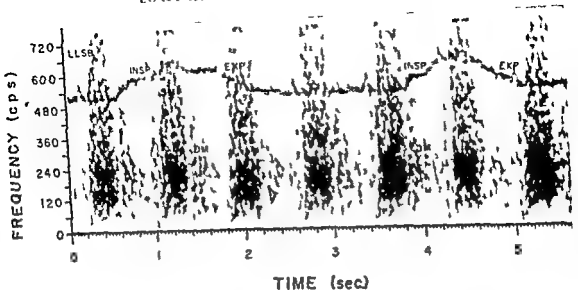


FIG. 371 VSD with aortic regurgitation

A 12-year-old white female was thought to have patent ductus arteriosus but none was found at operation. On re-examination it was concluded that the oxygen step-up on cardiac catheterization probably occurred in the outflow tract of the right ventricle rather than in the pulmonary artery.

As to whether it may be completely impossible to differentiate VSD and AR or PR from AS and AR.

A diastolic murmur of pulmonary regurgitation (e.g., Fig. 367) may occur. It was described in four of 19 cases of VSD in infants and children of ages from 3 months to 11 years (11). In some cases of VSD with pulmonary hypertension it is extraordinarily loud (see Fig. 369).

In the Eichmenger complex there is usually

little or no murmur attributable to the VSD. There is however likely to be a pulmonary early systolic click followed by decrescendo systolic murmur caused by dilated pulmonary artery and pulmonary hypertension.

The second sound with VSD particularly of the Roger type may be split to a moderate degree (Fig. 361). The mechanism of the splitting is probably the same as that in mitral regurgitation. The aortic valve closes before the pulmonary

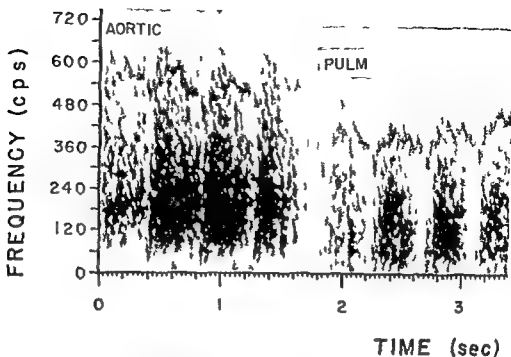


Fig. 370 VSD with aortic regurgitation

Aortic and pulmonary areas in 15 (A88576) 6 year old girl who was explored for patent ductus arteriosus but none was found. Cardiac catheterization findings were those of VSD. The blood pressure was 160/60 in the arms 200/60 mm Hg in the leg.

and high pulmonary blood flow. More often than not, however, these are concealed by the intense murmur generated at the defect(s). A pulmonary diastolic murmur may result from dilation of the pulmonary artery especially if pulmonary hypertension supervenes.

Most of the cases in whom closure of VSD by open heart techniques was deemed advisable have had a non holosystolic murmur, in apical diastolic rumble and some degree of pulmonary hypertension. In fact the incidence of the Carey Coombs murmur is much greater in such cases than in the classical case of *maladie de Roger* with holosystolic murmur. Despite increase in pulmonary artery pressure these patients must have high flow through the defect. Indeed, the high flow is probably responsible in large part, for the increase in pressure, there being little increase in pulmonary resistance. As far as reducing the length of the systolic murmur is concerned, it probably matters little whether the pulmonary hypertension is the effect of high flow or high pulmonary resistance.

Gasul and his colleagues (527, 528) have observed that a number of patients with VSD who previously had a loud, booming second sound in

the pulmonary area later had a definitely diminished  $P_2$ . Recatheterization demonstrated the phenomenon of acquired pulmonary stenosis in most of these patients. Hypertrophy in the infundibular area of the right ventricle had apparently developed and created a resistance to outflow from right ventricle to pulmonary artery.

The syndrome of VSD and aortic regurgitation (Fig. 370) (31, 68, 329, 735, 851, 1115, 1137 (p. 328), 1207, 1462 and 1592) can be mistaken for patent ductus arteriosus. It may also simulate the combination of aortic stenosis and regurgitation. Graphic differentiation is possible when the systolic murmur generated at the VSD is holosystolic and the decrescendo murmur of aortic regurgitation begins immediately with the second heart sound. A continuous murmur is created rather than the double murmur (with interruption after the systolic murmur) as in AS and AR. Furthermore, by graphic means the hope of the continuous murmur of VSD AR is more likely to be represented in Figure 368 than like that typical of PDA (Fig. 415). The same confusion may be created by VSD with pulmonary regurgitation. When the systolic murmur is not holo-

(Moss 19577) Serious cardiac dysrhythmias sometimes occur (270 179). About a third of involved persons are menorrhoeic. The diagnosis has been made in life by angiocardigrams which at the time of opacification of the left ventricle may show the aneurysm occasionally there is rupture of the aneurysm and development of the typical clinical and physiologic features of ventricular septal defect.

#### INFLUENCE OF THE SITES OF LESIONS

Ventricular septal defect occurs in of the ventricular septum and aneurysm of the coronary may have a common denominator all are congenital malformation of the ventro-ventricular septum (413). Sometimes aneurysm of a sinus of Valves is associated with one of the other two lesions (193 411 676 747). Syphilis can also cause such aneurysms they may occur with abscess bacterial endocarditis and with the Marfan syndrome and some cases have their origin in idiopathic cystic medial necrosis.

The right anterior coronary artery is most often affected. Ruptured aneurysm may be associated with a non specific systolic murmur and with the diastolic murmur of aortic regurgitation (1479). With rupture of the aneurysm into the right atrium, right ventricle or pulmonary artery the patient abruptly develops a continuous murmur with the plateau-decrescendo pattern shown in Figure 448. Typically the continuous murmur is distinguishable by its pattern from the friction rub-free-decrescendo double murmur of A-M but with more difficulty from the zigzag continuous murmur of IMA. It is difficult or impossible to distinguish it however from the murmur of ASD with AR other clinical features of it are based on the murmur of ruptured aortic aneurysm is often louder to the right of the sternum or over the mid and lower sternum—a feature which aids differentiation from the murmur of IMA.

#### CLINICAL CONSIDERATIONS

- (34) Pure pulmonary stenosis is valvular and infundibular pulmonary stenosis is with infundibular septal defect.

ANATOMICAL CONSIDERATIONS. There are two main anatomical varieties of pulmonary stenosis—val-

vular and infundibular. In addition an exceedingly rare supravalvular type is described (see below). Pure pulmonary stenosis is most often of the valvular type. Both types are present in the same patient. Among 20 patients Leatham and Westerman (544) concluded that valvular stenosis was present in 21 infundibular stenosis in 3, and both varieties in 1. The valvular type is fused together in a dome shaped diaphragm with a central perforation. The size of the orifice may be reduced to a fraction of a centimeter. In the infundibular variety of pulmonary stenosis the ridge like obstructing element is fibrous and muscular in nature and located in the vicinity of the crux subventricularis. Between the obstructing ridge and the normal pulmonary valve there is the so-called infundibular chamber (third ventricle of Kjellberg) which is walled by ventricular myocardium. Post-stenotic dilatation usually occurs in taking siphon in the pulmonary artery but probably only with the valvular form of the disease. Beck (1791) has written a monograph on the anatomy of congenital pulmonary stenosis.

HAEMODYNAMIC CONSIDERATIONS. The severity of pulmonary stenosis is an appreciable pressure gradient across the pulmonary valve (across infundibular zone). Elevation of right ventricular pressure and depression of pulmonary artery pressure are present. Peak systolic pressure in the right ventricle may exceed 200 mm Hg in extreme cases. Pressure is lower in the aorta is accounted for on the basis of the increased velocity of the blood at the stenosis and the large inertial kinetic energy as opposed to lateral pressure energy.

So-called relative pulmonary stenosis results from increased pulmonary blood flow as in ASD, VSD and TAV (see these entities). In total AR of the aortic valve type (287) a difference in systolic pressure across the pulmonary valve of as much as 12 mm Hg may occur on the basis of high flow alone. B. Wolff, Adams and Coodile (1120) found a 31 mm gradient in a case of ASD. Intertight. Contro. Miller and Derrick (287) could find no correlation between the volume of flow and the pressure gradient. This result suggested to them that another factor—presumably dilatation of the right ventricle and pulmonary artery—is operative.

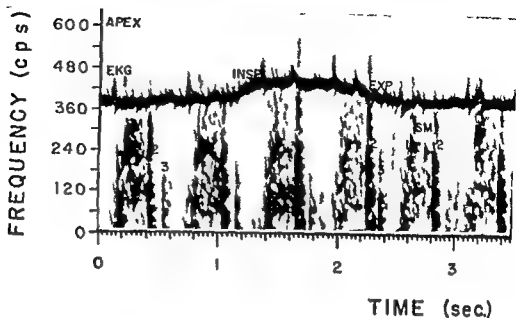


Fig 372

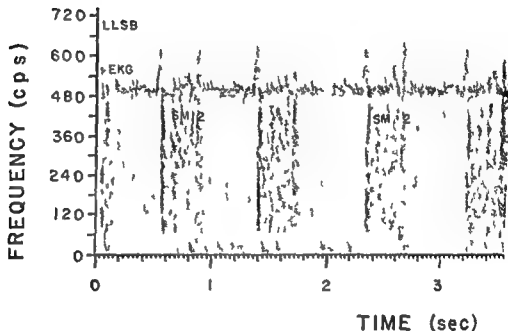


Fig 373

Figs 372 and 373 Differentiation of MR and VSD

A B (735323) 16 year old female was treated for SBI due to *Streptococcus fecalis*. Whether the underlying lesion was VSD or MR was not clear. At times the systolic murmur seemed loudest at the apex with third heart sound and possibly a short diastolic rumble. At other times the systolic murmur seemed loudest at the left sternal border. The SPC is consistent with either diagnosis since both could account for holosystolic murmur  $S_3$  and short diastolic murmur. The third sound/gallop is of left ventricular origin; it must underscore that relationship to the first (aortic) component of  $S_2$ .

probably because the left ventricle has two waves of ejection.

#### ANEURYSM OF THE MEMBRANOUS SEPTUM

Weakness may result in ballooning to the right of part or all of the membranous septum, usually

into the outflow tract of the right ventricle. A systolic murmur maximal at the left sternal border and suggesting ventricular septal defect or pulmonary stenosis, is present in some cases, absent in others (1438). The aneurysm may produce enough obstruction to right ventricular outflow to result in right sided heart failure.

\* See reference 231

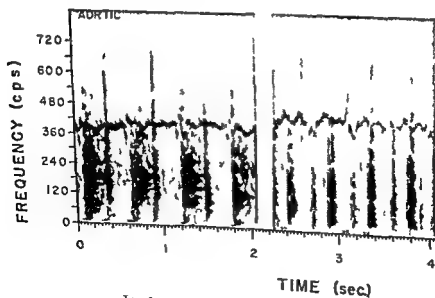
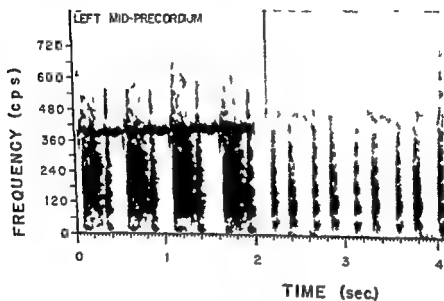
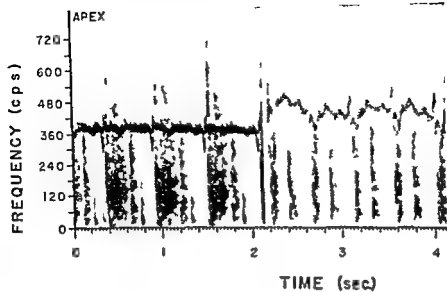


FIG. 34 (D E) See legend Figure 374 1

Anatomically, in cases of valvular PPS, it is difficult to imagine closure of the pulmonary valve in the usual sense. However, there appears to be a to and fro snapping of the diaphragm snapping upward with ventricular systole and snapping back toward the ventricle when pressure there falls below that in the pulmonary artery. Furthermore, pulmonary regurgitation of

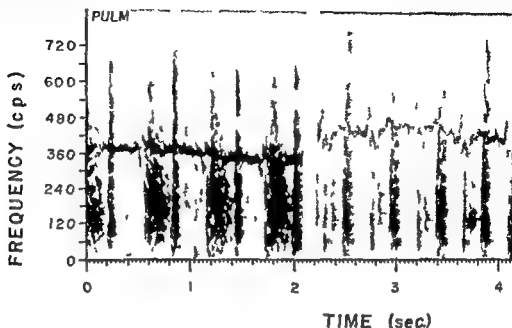


FIG 374 A Surgical closure of VSD

Recordings before (left) and after (right) operation on K. I. (742684) 8 year old patient in whom a large (12 by 18 mm) defect high in the right ventricular outflow tract distal to the crista supraventricularis was closed surgically. Pulmonary artery pressure was 65 mm Hg.

Before operation there was a decrescendo systolic murmur which stopped shortly before  $S_2$ —typical of the murmur in VSD with moderate pulmonary hypertension. In the pulmonary area there was an early systolic click distinct from  $S_1$  introducing the murmur. There was at the apex a protodiastolic gallop caused by hypervolemic filling.

After operation  $S_2$  was slightly split (right bundle branch block had appeared) the systolic murmur and gallop were gone. The early systolic click was seen unobscured by murmur (see IFSB) and there were systolic clicks probably of pericardial origin.

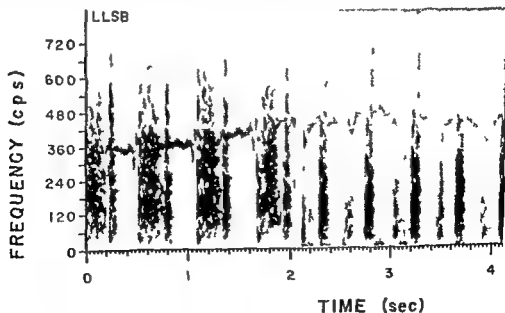


FIG 374 B See legend Figure 374 A

chamber in which the pressure curve has the contour of that in the ventricle but has the much lower systolic peak as that in the pulmonary artery. A simulating pattern which can be confused is produced if the catheter tip intermittently slips through a valvular stenosis with each systole.

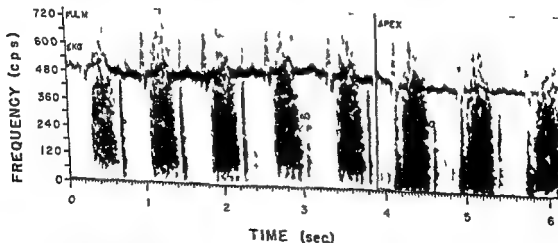
Rodbard and Shaffer claim (1960) that there are two varieties of infundibular stenosis: in one the stenosing ridge is largely fibrous and the degree of stenosis is unalterable; in the other type the myocardium constitutes a large portion of the obstructing ridge and through active contraction of this muscle the degree of stenosis is increased in the later part of systole. Angell and her colleagues (1958) present convincing evidence that in cases of pure valvular stenosis myocardial hypertrophy in the area of the infundibulum produces a dynamic stenosis in systole. This hypertrophy accounts for the failure of right ventricular pressure to fall to normal immediately after operation. They tend, however, to be a slow fall in right ventricular pressure to normal later after operation.

Cyanosis does not occur with IPS unless there is associated ASD. The atrial defect may be only probe (or flap) patency of the foramen ovale

with widening of the opening through increase in right atrial pressure.

Pulmonary stenosis is a state of systolic overload of the ventricle. The electrocardiogram shows the characteristic changes, i.e. marked right axis deviation and R waves of large amplitude in the leads at the right end of the precordial series, but lacking concentric hypertrophy of the ventricle.

CARDIOVASCULAR SOUND. The characteristic sys-



B

Fig. 3. Pulmonary area (B) in J. W. (193150) with pulmonary stenosis (infundibular type) and ASD. X-ray (A) shows extrasternal shadow on left hand border interpreted as dilated infundibular chamber—occulted third ventricle of Kjellberg (upper arrow) and elevation of the apex (lower arrow). The murmur can be accounted for entirely on the basis of the pulmonary stenosis, although it is possible that the ASD is contributing. The unusually loud but decaying pulmonary area sound is consistent with infundibular stenosis. The spectral pattern of the sound is such, however, as to suggest that the valve is not completely normal.



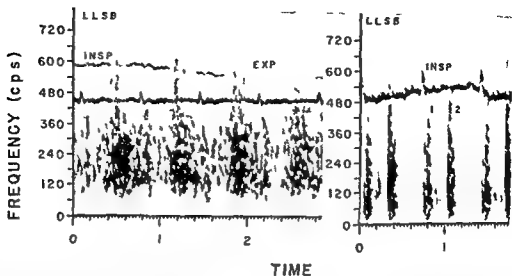


FIG 375 Ruptured sinus of Valvula occurred in D W (70600a) 33 year old woman early in the puerperium. The communication was between the posterior sinus and both the right atrium and right ventricle at the level of the tricuspid ring. The fistula was closed surgically. No diastolic murmur persisted after operation (right)

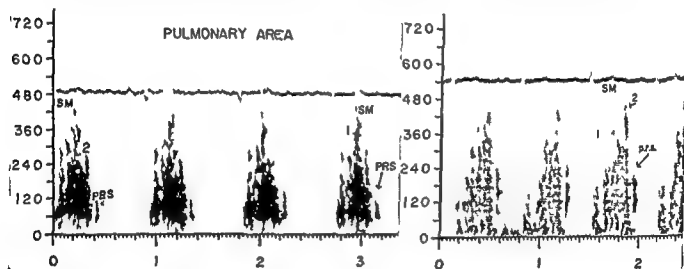


FIG 376 Valvular pulmonary stenosis

In each case the murmur extends to the aortic component of the second sound and there is a delayed relatively diminutive pulmonary component which might be called pulmonary reversional snap (PRS). This has the peculiar characteristics of a snap in that its frequency bottom does not extend to zero.

significant proportions is unlikely to occur because of the low pressure in the pulmonary artery. The ventricular snap of the stenotic diaphragm—which will be referred to as valve closure—occurs belatedly. One might guess that prolongation of the contraction of the right ventricle and the large discrepancy between right ventricular and pulmonary artery pressures both contribute to the delay in pulmonary valve closure. Iatham (see below) finds a close correlation between the level of right ventricular pressure

and the degree of delay in pulmonary closure as indicated by the pulmonary closure sound.

Silber and co-workers (1990) suggested a 'stenotic index' for semi quantitative estimation of the severity of pulmonary stenosis.

Infundibular stenosis can be differentiated from valvular pulmonary stenosis by the 'pull back' pressure curves obtained on withdrawal of the catheter from the pulmonary artery during cardiac catheterization. With infundibular stenosis pressure is recorded from an intermediate infundibular

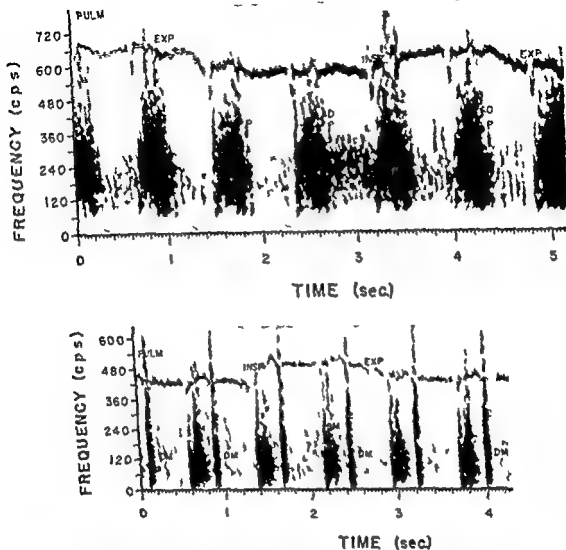


FIG. 3-9. Accentuation of Aortic murmur with inspiration.

Case 1 (20449) is a 10-year-old white male demonstrating clinical features of aortic-right ventricular hypertrophy. Postoperative dilatation of the aortic arch at the 1st sternal border (160). Before operation the aortic murmur was definitely accentuated by inspiration. The pulmonary stenosis valvelet in type was relieved under direct vision (below). Same case after operation. The pulmonary stenosis valvelet in type was relieved under direct vision. The type resulting mainly perhaps from residual dilatation of the pulmonary artery. No splitting of  $S_2$  was seen at any area. There is a faint early diastolic murmur.

pulmonary artery dilated in connection with the post-stenotic phenomenon. It is possible that both types of splits occur. Kjellberg et al (800) noted as did Leatham and Vogelstein (866) that the early systolic click occurred only in mild cases. Leatham and Weitzman (866) found what they considered to be a typical pulmonary regurgitation sound in all of 11 patients with very mild pulmonary stenosis and cases with a normal

electrocardiogram and a right ventricular pressure of 40 mm Hg or less. The sound in LPS was earlier (at 0.05 sec after  $S_1$ ) than in cases of pulmonary hypertension with dilated pulmonary artery (at 0.07 sec). However in 33 cases of a more severe variety only five showed the sound and the child right ventricular pressures among the lowest values for the group. In one patient the sound appeared after a thorotomy.

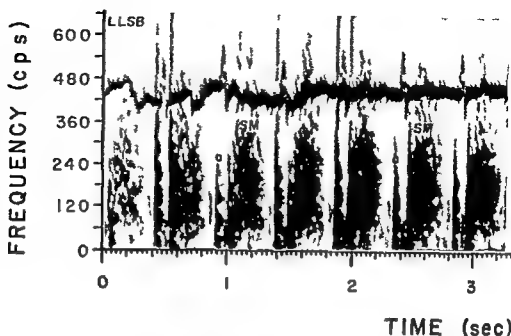


FIG. 378. Pre-systolic gallop with pulmonary stenosis.

LLSB in J S (652901) is a 1-year-old female with pure pulmonary stenosis of combined infundibular and valvular types. Congenital ptosis was an associated anomaly. The atrial sound is the loudest sound in this area. A diminutive sound just before the atrial gallop is probably a much delayed pulmonary closure sound.

*tolic* murmur of pulmonary stenosis is an ejection stenosis murmur like that of aortic stenosis. In the o-cillographic PCG it is diamond-shaped, in the SPCG it has the configuration of a Christmas tree. In general, the peak of intensity and frequency of this murmur is later after the first heart sound (or QRS) than in the case of the murmur of aortic stenosis. However, as pointed out by Leitham (862) just as in aortic stenosis in which a silent gap separates the end of the murmur from the aortic closure sound, there is in pulmonary stenosis a gap between the end of the murmur and the delayed pulmonary closure sound, even though the murmur usually extends up to the aortic closure sound.

Vogelpool and Shirre (1493) showed that it is possible to differentiate pulmonary stenosis with intact ventricular septum from cases with VSD as in tetralogy of Fallot, in the latter case the murmur usually stops before the aortic closure sound. In the former case, as just stated, the murmur extends at least to—and usually beyond—the aortic closure sound. The differentiation is particularly useful in connection with pulmonary stenosis and ASD with reversed shunt, which may simulate tetralogy of Fallot. In the

experience of Leitham and Weitzman (860) however, only 7 of 26 cases of tetralogy showed a murmur which stopped before the aortic closure sound. It can at least be stated that when the murmur does display this pattern, tetralogy can be diagnosed.

The murmur of PPS is maximal in the pulmonary area. It may radiate toward the left shoulder and to the back, but usually not to the base of the neck on the right as does the murmur of aortic stenosis, although when very loud it may even be heard in that area. In valvular stenosis the murmur and thrill are likely to be detectable in the suprasternal notch and maximal in the second left interspace. In infundibular stenosis the murmur is often maximal somewhat lower—in the third interspace.

The murmur is likely to be introduced by a snap (described first by Petit (1204)) which is too late in relation to the QRS to be first heart sound<sup>7</sup> and is interpreted as being caused by the abrupt doming of the stenotic valve diaphragm into the pulmonary artery. Another possible mechanism is snapping of the wall of the pul-

<sup>7</sup> It may be this snap which has led some (1205) to conclude that the first sound is accentuated in PPS.

with the production of slight paradoxical splitting of S in extreme instances. However, the prolongation of left ventricular systole never attains the marked degree seen with pulmonary stenosis. The left ventricle by design a more satisfactory pressure pump is better able to cope with the increased burden. The right ventricle by design primarily a volume pump (1323) however quantitatively different reaction to outflow obstruction.

Prolongation of right ventricular systole cannot be attributed (846) to delay in the onset of right ventricular systole since this averaged 0.07 sec from the beginning of the Q wave a figure agreeing with the finding of Coblenz et al (276). Nor is there any abbreviation of left ventricular systole which averaged 0.11 sec a normal value in cases of pulmonary stenosis. Right ventricular systole averaged 0.79 sec (range = 0.45-0.91 sec).

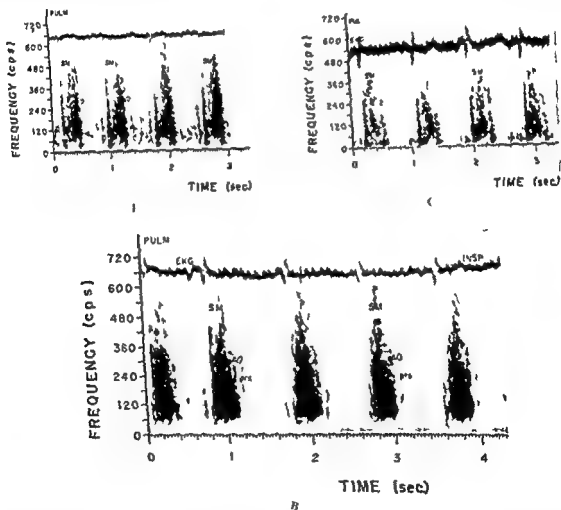


FIG 381 Valvular pulmonary stenosis with VSD

Transventricular valvulotomy was performed soon after the recording shown (above left). The patient is one of the 10 per cent which has no pulmonary reversal sound. (Most of the sound in diastole is thought to be of non cardiac vascular origin.) At operation the anatomy seemed to be typical of valvular stenosis. The dome like stenosis would be 1/2 inch. The right ventricular muscle was very thick. After the valvulotomy pressure in the right ventricle was 110/50 mm Hg systolic. In the recording taken 3 months after operation (bottom) there is still a pulmonary systolic which however has an earlier peak. A delayed pulmonary reversal sound (pr) is now present. 14 months after operation the murmur (above right) had greatly diminished.

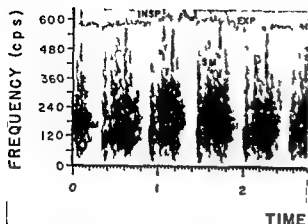


FIG 380 Pulmonary stenosis and regurgitation. Pulmonary area in N. T. (735272) asymptomatic 13 year old female. Cardiac catheterization showed a right ventricular pressure of 40/5 mm Hg and mean pulmonary artery pressure of 8 mm. The main pulmonary artery was dilated and pulsatile. EKG showed slight RAD and RSR pattern in  $V_1$ . The split S characteristic of pulmonary stenosis is present. The pulmonary component is less delayed or muffled than in more marked cases. The diastolic murmur is probably lower pitched than is the case with aortic lesions.

There may be an appreciable delay (Fig 377) between the first sound and the onset of the murmur (706, p 143). Münheimer (1033) suggests that ballooning of an infundibular chamber in valvular PPS may be a factor in this delay. He proposes the presence of the  $S_1$  murmur gap as a point differentiating valvular from infundibular PPS. He (1033) thought that the peak of the murmur of infundibular stenosis occurs earlier. Kjellberg *et al* (800) thought that there are other features which permit differentiation in infundibular stenosis: the murmur usually extends only as far as the aortic closure sound whereas in valvular stenosis it is likely to extend beyond it because of the late contracting infundibulum. Vogelpoel and Shrire (149) also thought differentiation was possible. However, Lenthum and Weitzman (866) concluded that there are no differentiating points.

Frequently, after operation for pure pulmonary stenosis of the valvular type a considerable systolic murmur persists despite the fact that the operative procedure was thought to have been technically satisfactory. The explanation is probably provided by the observations of Ingale and colleagues (428) in such cases there is often

hypertrophy in the infundibular area of the right ventricle and a dynamic type of outflow obstruction. After several months this factor disappears. Figure 381 presents the records of a patient with pure pulmonary stenosis who still had a considerable murmur 4 months after operation but had lost it almost completely 14 months after operation.

Unlike that of true pulmonary stenosis, the murmur of relative pulmonary stenosis is limited to early systole. In organic pulmonary stenosis the delay in the peak of the systolic murmur results from a change in the pattern of right ventricular contraction.

The pulmonary closure sound, perhaps better termed pulmonary reversal snap (1082), is always diminutive (except in the mildest cases) and is delayed. Its intensity is, however, a poor index of the severity of the pulmonary stenosis, just as the degree of depression of pulmonary arterial pressure is a poor index. Variability in the intensity of the malformed valve, in the proximity of the pulmonary artery to the chest wall, in the thickness of the chest wall and possibly in other factors is too great to permit assignment of quantitative significance to the intensity of the pulmonary reversal sound. On the other hand the interval between the aortic and pulmonary closure sounds has been found to bear a fairly direct relationship to the degree of elevation of right ventricular pressure (Fig 382). This relationship no longer obtains when patent ductus arteriosus or VSD is associated. However, the association of ASD does not vitiate the relationship.

Although there is close correlation between right ventricular pressure and the interval between the aortic and pulmonary components of  $S_1$  it is not necessarily correct to assume that the delay of the pulmonary component is caused merely by the fact that a longer time is required for the high pressure in the right ventricle to fall below that in the pulmonary artery. It is likely that the pattern of right ventricular contraction is seriously altered with prolongation of systole as an important feature. The peak of the systolic murmur is later in systole than is the case with the murmur of aortic stenosis. The aortic closure sound may be somewhat delayed in aortic stenosis,

Although the systolic murmur of pulmonary stenosis is reduced in duration and intensity after operation it is never abolished completely even by direct valvotomy. Following operation for valvular pulmonary stenosis the systolic murmur may subside slowly, presumably because an element of infundibular stenosis on the basis of myocardial hypertrophy is reversible.

Atrial gallop occurs frequently (3/109) with pulmonary stenosis, with other instances of systolic overload of the ventricle. Laubry and Peszy (848) and Blackford and Parker (109) emphasized its audibility over the cervical vein. However, Leatham and Weitzman (866) found an atrial sound in only 2 of 33 cases of moderate and severe cases of PLS. An auricular systolic murmur of low intensity occurred in three patients who displayed a prominent *a* wave in the venous pulse of the neck. They (866) speculated with some evidence in support that with atrial systolic blood might actually be forced through the stenotic area. Pelative *tricuspid stenosis* is an alternative explanation.

#### TETRALOGY OF FALLOT (T/F)

(Syn. Triad of Fallot)

**DEFINITION.** The complex of VSD, PS and overriding or dextroposed aorta constitutes the triad of Fallot. Hypertrophy of the right ventricle completed Fallot tetralogy but obviously is not a congenital malformation in the sense that the others are but rather a positional development in compensation for the malformation.

The designation *triad of Fallot* and *triad of Fallot* are used especially by Fallot's countrymen. The point raises legitimate objections to the eponym. Triad of Fallot is pure pulmonary stenosis with patent interatrial septum. The simulation of T/F is discussed below. Identical of Fallot is T/F plus patent interatrial septum. Clinically patients with this combination behave differently from patients with pure T/F.

**HAEMODYNAMIC CONSIDERATION.** (198-749, 1409) The obstruction to normal right ventricular outflow is usually infundibular pulmonary stenosis and less often valvular pulmonary stenosis. In combination of these two or pulmonary artery stenosis, older patients coming to autopsy tend to have valvular stenosis which is more severe than the infundibular stenosis.

(40) In younger patients infundibular stenosis predominates. Most valvular cases have infundibular stenosis of some degree as well.

The pulmonary valve beyond an infundibular stenosis is frequently bicuspid in the syndrome. The bronchial arteries become dilated and resist although inadequately pulmonary blood flow. Poststenotic dilatation of the pulmonary artery occurs only in a minority of the cases as might be expected because in most infundibular stenosis dominates. Usually there is hypoplasia in the region of the pulmonary artery and the cardiac apex is tilted up through enlargement of the right ventricle and possibly branching of the left. The net result is a slight hypoplastic valvular stenosis. Occasionally the pulmonary valve is totally absent the pulmonary stenosis is infundibular in type and an aortic diastolic murmur is added to the other auscultatory sign of T/F.

The aorta, rather than descending, is in the right and then to the left in its course in the anteroposterior direction tend to project straight cephalocaudal in the sagittal plane. Pittman and Emanuel (1184) have emphasized enlargement of the ascending aorta in T/F. The aorta narrows sharply abruptly in the vicinity of the isthmus.

The dilated bronchial collateral which may carry more blood to the lung than the normal pathway or may be the only route for pulmonary blood flow in cases of pulmonary atresia have been beautifully demonstrated by Hales and Lubow (621). They may be a striking isodisplacement of the esophagus and be otherwise demonstrable on radiologic study (290-291). Even notching of the ribs may occur through dilatation of the intercostal arteries, a part of the collateral circulation.

**PHYSIOLOGIC CONSIDERATIONS.** The basic physiologic defects in Fallot's syndrome are (1) obstruction to flow of blood to the lungs and (2) right to left shunt. The cyanosis, polycythemia and clubbing are well accounted for on this basis. Because of the overriding aorta there is less obstruction to ejection of the right ventricle into the systemic circulation than there is in simple VSD. The pressure in the right ventricle is elevated and at times the right ventricle becomes essentially the systemic ventricle.

Shunting has been emphasized by Tur

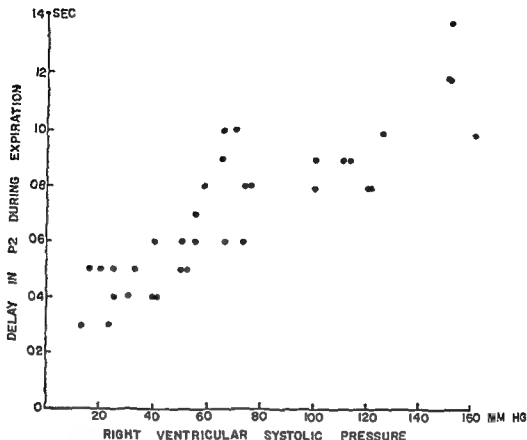


FIG. 382 The relationship between right ventricular pressure and the interval separating the aortic closure sound from the pulmonary reversal snap (From Leatham and Weitzman (866)).

Phonocardiographically, the delayed pulmonary closure sound (or pulmonary reversal snap) is demonstrable in the overwhelming majority of cases—about 85 per cent of cases of pure pulmonary stenosis (866). When not demonstrable, unusually severe pulmonary stenosis is likely to be present. The sound may appear after valvotomy in such cases and if previously present moves closer to the aortic component after operation. Stethoscopically Burritt (39) found a 'split second sound' in 11 of 33 cases. Abrahams and Wood (3) described a split  $P_2$  in 12 of 19 cases of mild stenosis, but in all severe and moderate cases  $P_2$  was unitary and of presumed aortic origin. In regard to the pulmonary closure sound there seems to be no difference between valvular and infundibular stenosis.

Kjellberg *et al.* (800) described a bizarre case in which the obstructing lesion was a diaphragm with central perforation located in the first portion of the pulmonary artery with a normal pulmonary valve and infundibulum below. In this

case, three sounds were present in the general vicinity of the normal second heart sound. These were interpreted as aortic closure sound, pulmonary closure sound and reversal snap of the monilous diaphragm. Williams, Lange and Hecht (1564) have observations on cases of supravalvular pulmonary stenosis.

Pulmonary regurgitation (Fig. 380) occurs rarely in congenital pulmonary stenosis. Leatham and Weitzman (866) found it in one case of valvular stenosis. The low pulmonary artery pressure probably militates against such a murmur. It is likewise rare for pulmonary regurgitation to develop after valvotomy, although such occasionally is observed. It developed in 8 of 22 patients in whom open valvuloplasty was performed by Swan plus all three of the patients in whom a segment of the valve was excised (120). It has also been encountered after transventricular valvulotomy (226). Pulmonary regurgitation is, however, a benign lesion (423, 476) when pulmonary hypertension is not present.

## CONGENITAL CARDIOVASCULAR DISEASE

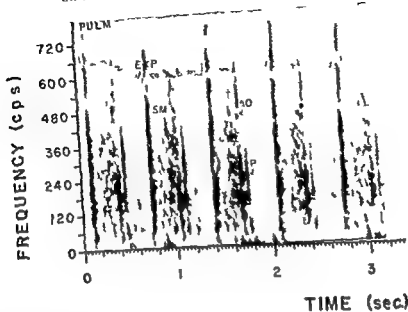


Fig 38A Pulmonary stenosis with ASD

Cly (no 30) 12 year old female had a clinical picture consistent with T/F. The sound are in the present with 18 (with ASD also) a diagnosis confirmed by cardiac catheterization. Contrast with characteristic pattern of T/F in Fig 38A and last half of 38B.

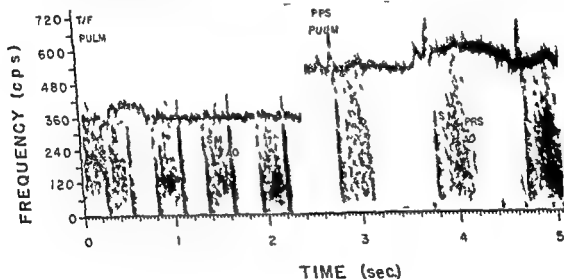


Fig 38B Tetralogy of Fallot compared with pure pulmonary stenosis

On left pulmonary stenosis (no 31) 12 year old girl with typical clinical picture and surgical findings of T/F. Characteristically systolic and in most cases the systolic murmur is slightly before it. On right tetralogy of Fallot compared with pure pulmonary stenosis.

Intensity of the systolic murmur occurs after exercise especially after exercise with tending— situation in which flow across the stenotic pulmonary valve is reduced and flow through the ASD increased. A rare but physiologically inter-

esting situation which is the converse of T/F and exercise. T/F combined with coarctation of the aorta in such cases the systolic murmur of T/F is louder and longer and a pulmonary closure sound may be present. The increased systolic



sig (1160), a characteristic posture which patients with tetralogy of Fallot, and less commonly with other varieties of cyanotic congenital heart disease, assume after exertion. The child begins to display the phenomenon when he starts to walk. Social pressures may lead to its disappearance at the age of eight to ten years. Thereafter, crossing the legs with entwining and squeezing may take the place of squatting. Arterial oxygen saturation falls during and after exercise but recovery is more rapid with squatting (Fig. 386). Fall in systemic resistance (99%) through peripheral vasodilatation with constant resistance to pulmonary blood flow results in fall in pulmonary flow. The changes in flow relationships can be well demonstrated by the analogy of an electrical model with two parallel resistances, one of them variable.

McCord and Elk and Blount (1038) make the cogent observation that the tetralogy of Fallot encompasses a broad spectrum of physiologic changes and a correspondingly broad clinical spectrum. In the opinion of these workers pulmonary stenosis and ventricular septal defect are the essential lesions and dextroposition of the aorta of secondary importance. They present evidence that there is not always equilibration of pressure in the two ventricles. Because of the variability, L/T may masquerade as VSD with

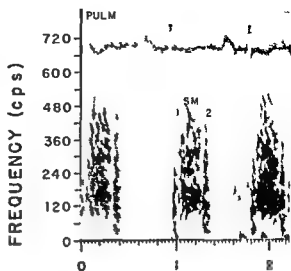


FIG. 384. Aortic area in J S (16075r) 43 year old female with clinically typical tetralogy of Fallot established by cardiac catheterization. Rather extreme hypertrophic pulmonary osteoarthropathy, 1 prenatally. Systolic murmur topping slightly before S<sub>2</sub> as in aortic stenosis. Contrast characteristic pattern with that of pure pulmonary stenosis (e.g. Fig. 316).

left to right shunt or as isolated pulmonary stenosis.

I further light is shed on the interrelationship of T/I of the cyanotic and non cyanotic types (1318) with simple VSD by the findings of Gisul and colleagues (528). When infants who were found on first catheterization to have changes of simple VSD are recatheterized evidence of a gradient from right ventricle to pulmonary artery and even reversal of the direction of the shunt may be found. This probably explains the fact that striking improvement in c/c's of VSD often occurs after the first year of life. Possibly the mechanism of the "required" pulmonary stenosis is hypertrophy of the crista supraventricularis.

CARDIOVASCULAR SOUND. The systolic murmur is probably produced (149a) mainly or exclusively at the stenotic pulmonary outflow area (Fig. 383). Because of the overriding of the aorta there is probably little obstruction to outflow through the aorta; therefore a murmur is not likely to arise at the VSD. Supporting the view that the pulmonary stenosis is the generator of the murmur are the facts that (1) in pulmonary atresia there is little murmur, (2) with thrombosis of the outflow tract which occasionally develops a pre-existing murmur disappears, and (3) a decrease in the

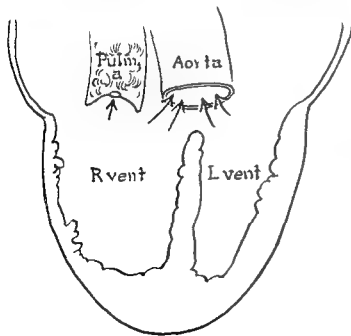


FIG. 383. The murmur of T/I is usually generated at the pulmonary stenosis.

TABLE 15

The differentiation of tetralogy of Fallot from tetralogy of Fallot (pulsus parvus et irregularis) with patent interatrial septum

| Important Difference                        | Tetralogy of Fallot  | Tetralogy of Fallot with Patent Interatrial Septum   |
|---|--|--|
| <b>Sound</b>                                |  |  |
| Systemic murmur                             | Step before aortic component of $S_2$  | Extends to aortic component  |
| Pulmonary component of $S_2$                | Usually absent   | Usually present in delayed and diminished form (pulmonary reversal flow)   |
| First ejection sound (early systolic click) | Frequently present originates in aorta relatively late in systole called wide splitting of first sound                     | Present in mid systole only originates in pulmonary artery relatively early in systole called closure splitting of first sound |
| Aortic component of $S_2$                   | Often accentuated in pulmonary area  | Usually unchanged  |
| <b>ECG</b>                                  | Extreme right ventricular hypertrophy and strain rare  | Extreme right ventricular strain may occur   |
| <b>Venous puls</b>                          | Giant a waves never present<br>Dominant m waves unusual  | Dominant or giant m waves the rule   |
| <b>Fluorograph</b>                          |  |  |
| Heart size                                  | Usually not increased  | May be increased   |
| Upper part aorta                            | Small  | Full from post-tensionic dilatation  |
| Aorta                                       | Wide right aortic arch in 20%  | Relatively normal right arch very rare   |
| Heart to spine                              | Slightly elevated apex convexity of mid left heart lordosis  |  |
| <b>Inspection</b>                           | $S_2$ often palpable in aortic area often systolic thrill in pulmonary area from anteroposterior aorta thrill often absent | Unchanged left over right with a tricuspid thrill usually present  |
| <b>Cyanosis</b>                             | May develop late but usually before 14 months  | Usually develops late  |
| <b>Swelling</b>                             | More common (about 50%)  | Occurs less often (about 10%)  |
| <b>Angina pectoris</b>                      | Very rare  | Extremely common   |

Adapted from data of Vogelpoel et al (1971) and Campbell (1971)

in the extreme cases in which it is a systemic ventricle must begin to relax before the aortic valve can close. The murmur generated at the stenotic pulmonary valve will cease before the aortic valve closes. One might think of this relationship in terms of pressure and murmur threshold. The threshold of pressure for generation of murmur is likely to be higher than the threshold for opening (and closing) of the aortic valve. With the rise of intraventricular pressure the aortic valve will open before the onset of the murmur, with fall in intraventricular pressure the murmur will cease before the aortic valve closure.

The fact just stated is useful (1971) in differentiating T F from T P with right-to-left shunt at the atrial level (compare Figures 28 and 14). Other points helpful in this differentiation are presented in Table 15. Two reports (SGG 1976) which appeared almost simultaneously are in agreement on most of the descriptive aspects of T F as opposed to T P with interatrial communication—a possibly confusing problem in clinical differential diagnosis (1970). However they disagree on how characteristically of each condition is the duration of the systolic murmur. Thus scrutiny of the cases of Vogelpoel and co-workers (1976) appears to indicate that the systolic mur-

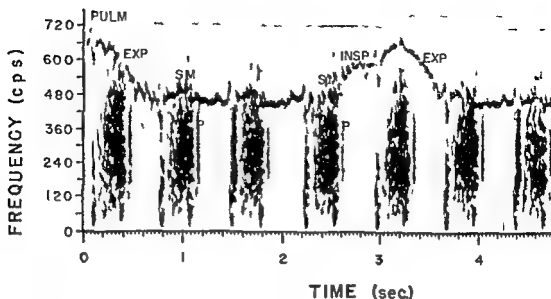


FIG 385C Valvular pulmonary stenosis

Pulmonary area in M K P (773401) 14 year old girl confirmed at operation Right ventricular pressure was 137 mm systolic The characteristic late peaking of the systolic murmur and the diminutive pulmonary reversal sound are well demonstrated

resistance seems to favor pulmonary flow Clinically, the patients do better than do patients with only T/T of similar anatomical type (4) Finally, there is a rather satisfactory correlation between the volume of pulmonary valve flow and the intensity and duration of the systolic murmur of pulmonary stenosis In pulmonary stenosis with intact septum pulmonary valve flow has in the experience of most laboratories been approximately twice that in cases of T/T

Sometimes, because of the fact that the aorta is carrying almost all, or in fact all of the cardiac outflow a murmur of high flow is generated in the aorta An early systolic click (ejection sound) arising in the aorta may occur for a similar reason (989) The ejection sound usually occurs about 0.07 sec after the first sound It is most likely to occur after Blalock Pinnag or Pott's procedure, because flow through the aorta is augmented The ascending aorta is likely to be closer to the sternum than normally, this favors transmission of sounds generated in the aorta Furthermore, the enlargement of the ascending aorta emphasized by Pittinson and Emanuel (1185) may be a factor in the systolic click That the sound does not arise in the pulmonary artery is attested by its presence in cases of T/T with

pulmonary atresia Furthermore the usual lack of dilatation of the pulmonary artery makes this structure an unlikely source of the sound Still further Leatham and Weitzman (866) observed a case in which the murmur (arising presumably at the pulmonary stenosis) began before the sound (arising presumably in the aorta)

Although the murmur of iliot syndrome is generated at the pulmonary stenosis it usually differs from that of PPS It is variable in its intensity from case to case It may stop with a brief silent gap before the aortic closure sound (e.g. Fig 384), rather than running up to the aortic closure sound as in the case of the murmur of PPS The peak of the Christmas tree murmur occurs earlier in systole The basis for the difference in the heart sound pattern of T/T from that of uncomplicated PPS is not the fact that most PPS is valvular where is infundibular stenosis predominates in cases of T/T Rather the VSD of T/T is fundamentally responsible for the difference In aortic stenosis the systolic murmur stops slightly before the aortic closure sound because obviously contraction of the ventricle must relax a bit before the aortic valve can close and the murmur will cease with relaxation of the ventricle In I/I the right ventricle especially

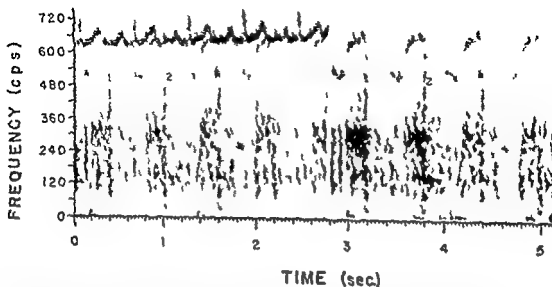


FIG. 25. Mitral murmur from tracheal collateral in T/t

Right infraplavine for area in A & V (a) 6 year old female with typical tetralogy of Fallot except for the aortic valve element consisting of a single orifice which divides into an arterial pulmonic valve and a right ventricular valve which are fused to the pulmonary valve. The right ventricle is dilated and the pulmonary valve is stenotic. The right ventricle is dilated and the pulmonary valve is stenotic. The right ventricle is dilated and the pulmonary valve is stenotic.

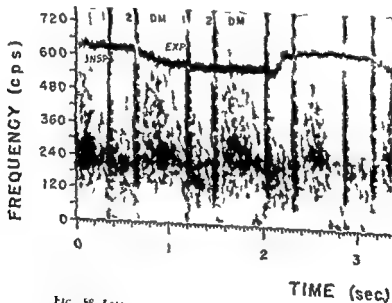


Fig. 58 Late complication of Blacklock Tau in T/t

[illegible]

mur followed the pattern outlined above in 13 of 14 cases of PPS with interatrial communication and 39 of 48 cases of T/F. Leatham and Weitzman (866), on the other hand found that the systolic murmur stopped before the aortic closure sound in only 7 of 26 cases of T/F.

Vogelpoel (1495) thought the pre-systolic murmur which occurs in some cases of PPS and the loud atrial sound which occurs in others might be helpful in differentiating T/F from PPS with ASD. However, Leatham and Weitzman (866) found an auricular systolic murmur in two patients with T/F and large a waves in the neck.

In T/F the second heart sound tends to be

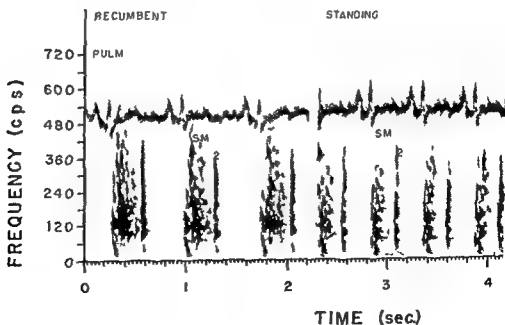
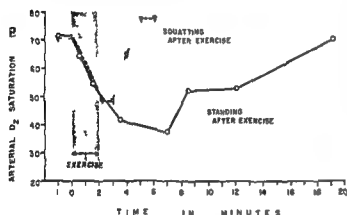


Fig 386 (160c) In T/F the effect of exercise with and without squatting on the oxygen saturation of arterial blood (from Lurie (998)). (Below) Decrease in murmur of T/F when the patient (10 K 370939) is standing is compared with the recumbent position. Recordings made with identical amplification. The effect of exercise followed by quiet standing on the murmur of T/F in the same patient.

unitary—exclusively aortic closure sound. So called “P” may be normally loud or possibly even increased. The aortic closure sound is usually well heard in the pulmonary area because of the anatomic reorientation of the aorta. Furthermore, unlike PPS, in which a diminutive, delicate pulmonary closure (or reversal) sound is demonstrable, at least by graphic means, in about 80 per cent of patients, in Fallot’s syndrome the incidence is about 15 per cent. Vogelpoel and Schrire (1495) found it in 3 of 18 patients. When present it should be suspected that pulmonary flow is greater than the average. Clinically, the patients are less misperceived. The sound may appear after Blalock-Taussig operation or direct repair of the outflow tract. Leatham and Weitzman (866) describe an instructive patient in whom the pulmonary closure sound first appeared after Blalock-Taussig operation, located 0.11 sec after the aortic component after subsequent valvotomy, the interval was reduced to 0.06 sec and an early diastolic murmur appeared following the pulmonary sound and displaying a crescendo-decrescendo pattern.

Vascular bruits sometimes continuous, presumably arising in the bronchial artery collaterals.

tensio and tricuspid stenosis need no particular elaboration. Congenital tricuspid stenosis can occur as an isolated valve lesion unlike the rheumatic form of the disease which almost always is accompanied by disease of the mitral and/or aortic valves. Essential to survival in tricuspid stenosis is the existence of an interatrial communication (so that venous blood can have access to the left side) and usually a VSD for transportation of blood to the lungs.

**PHYSIOLOGIC CONSEQUENCES.** In the Ebstein's malformation there is the anomalous functional situation such that part of the wall of the pretricuspid chamber contracts at the same time as does the ventricle. Because of the decrease in the ordinary right ventricle there is impairment in the propulsion of blood into the pulmonary circulation. Furthermore, there is circumstantial evidence from cardiac catheterization as well as from studies of the murmur that contraction of the ventricular component of the pretricuspid chamber contributes to right-sided ejection. Specifically pressure curves from the pulmonary

artery show a secondary rise in the latter part of the period of ventricular systole synchronous with a similar rise in pressure in the pretricuspid chamber (108, 119).

Cyanosis may be present if there is an interatrial communication. The EKG shows large notched P waves and there is usually pulsation of the neck veins and liver. A left parasternal heave is conspicuous by its absence. In the diagnosis simultaneous intracardiac EKGs and pressure tracings by means of a double cardiac catheter may be helpful for a reason which will probably be obvious although usually the other features listed here are adequate for a satisfactory clinical diagnosis and cardiac catheterization may be hazardous (1, 100). Often there is rather a remarkably good exercise tolerance in relation to the size of the heart, the impregnateness of the gallop, and the degree of cyanosis. Survival may be long, to the age of 79 years in one case (7). Recognized with increasing frequency in adult Ebstein's anomaly is a variety of congenital malformation which is frequently mistaken for rheu-

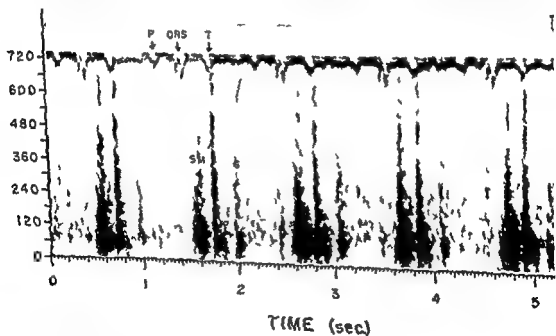


FIG. 300. Ebstein's anomaly.

Lead I in left midprecordial in 14 year old male. G. R. (1951). Right bundle branch block and prolonged P-R interval are present. The first count (1) is fast. There is a mid systolic murmur (SM) with the configuration of a systolic ejection murmur. The second component of the split S and S<sub>1</sub> probably pulmonary flow (1) is diminished relative to the first (1). A protosystolic gallop (C) is present and probably initiates a short murmur.

to the lungs, may be audible. They are often so striking as to suggest the diagnosis of patent ductus arteriosus. Occasionally these bruits are musical (see Fig. 387).

The creation of an artificial ductus arteriosus by the operation of Blalock and Tussig or that of Potts results in a continuous murmur with the graphic configuration characteristic of mitral regurgitation. Abbreviation or disappearance of this murmur is a clue to the fact that obliteration of the anastomosis is occurring. More and more patients in whom Blalock-Tussig or Potts operations were performed ten or more years ago are having return of cyanosis and disability. In many of these criteria other than the murmurs must be used in evaluation of the original anastomosis because the murmur of bronchial collaterals may be indistinguishable from that of the anastomosis.

A pulmonary early diastolic murmur may develop after operation. Such is more common after direct operation but interestingly may occur

after shunt operations, particularly if an excessively large shunt is created with subsequent dilatation of the pulmonary artery (Fig. 388).

### TRICUSPID LESIONS

(including Ebstein's malformation, tricuspid stenosis (900), tricuspid atresia)

**ANATOMIC CONSIDERATIONS (1416)** Ebstein's malformation (398, 410, 792, 1091) consists of a downward displacement of the tricuspid valve such that part of the right ventricle becomes incorporated into the right atrium, or, at least, into the pre-tricuspid chamber. The tricuspid valve is usually deformed in the way of fenestration, double orifice (800), etc., so that one would from the necropsy appearance, anticipate both regurgitation and some obstruction to forward flow. In about half of all cases there is an associated interatrial communication. The pre-tricuspid chamber becomes huge. The pulmonary artery, on the other hand, is usually small.

Description of the anatomic site in tricuspid

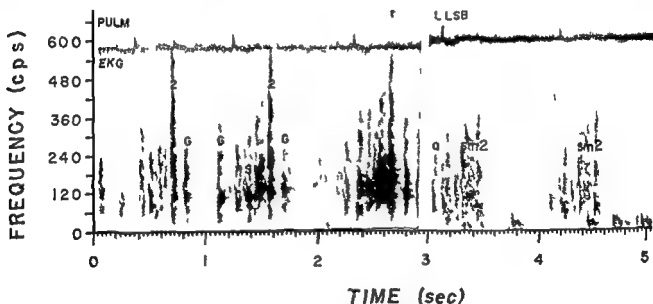


FIG. 389 Ebstein's malformation

R J T (A91519) 11 year old white male has from birth been cyanotic and dyspneic on exertion with "quitting" and fainting spells. Angiocardiogram at age of 7 showed very large and slowly emptying right atrium with large ASD. I K C reveals large P wave with PR of 0.28 sec. and right bundle branch block. The PR interval became normal after the longer diastolic periods of sinus arrhythmia.

SPC (S) There are both presystolic and protodiastolic gillops and a mid or late systolic murmur which has the appearance of an ejection systolic murmur and which may be caused by forward movement of blood through the tricuspid valve. The atrial gillop is at times a short murmur and because of a long IR interval is usually in mid diastole. In the last cycle (pulmonary area) the 1P is shorter after a longer diastolic pause and the atrial sound is closer to the first heart sound. In some areas the protodiastolic sound is snapping as indicated by its pure frequency content at about 200 cps. This is probably caused by snapping of the tricuspid apparatus in early diastolic inflow.

**CARDIOVASCULAR SOUND** Among 82 patients with the Ebstein malformation reviewed by Kilby *et al* (792) there were only 30 without murmur, 11 aortic and/or diastolic. The second important feature (and among congenital malformations a feature rather characteristic of the Ebstein malformation) is the presence of trilling diastolic gallop sound. These were present in 30 of 82 patients (792). Often a quadruple rhythm results from the presence of both third sound and fourth sound gallop. Although more will not be said about the gallop sounds, it should be emphasized that they are perhaps the most important auscultatory feature of the Ebstein anomaly.

(1) **Aortic murmur** almost always present. Often it has been attributed to tricuspid regurgitation. The anatomic change in the aortic and the graphic demonstration of a holosystolic murmur (Fig. 392) are in accord. This murmur is likely to be audible at the usual position of the cardiac apex because the tricuspid valve is situated further to the left than is ordinarily the case.

At other times the aortic murmur is circumcribed and late and has the configuration of inflection tone murmur. This murmur may result from contraction of the ventricular myocardium in the wall of the pretricuspid chamber with forcing of blood through the malformed tricuspid valve. See Figures 390 and 391 for an ECG of this phenomenon and see Figures 499 of Kjellberg *et al* (600) and Figures 1 and 3 of Kilby *et al* (792) for oscillographic displays of the essentially identical phenomenon.

In still other cases the sound in systole is more like a sound (in the usual sense) than a murmur. This sound may likewise be in the firstarily caused by contraction of the ventricular component of the wall of the pretricuspid chamber. In essence it may be an atrial heart sound occurring in ventricular systole because of the particular anatomic and physiologic relationship of Ebstein's malformation.

(2) Usually the diastolic murmur(s) of the Ebstein anomaly similarly have origin at the malformed tricuspid valve. The murmur may be mid-diastolic or more often presystolic (i.e., atrial) and usually be heard at the left sternal border. It is appropriately referred to as an atrial murmur since the PR interval is often long and the mur-

mur not strictly presystolic. It was present in five of eleven cases at the Mayo Clinic (792). Five authors have presented phonocardiograms showing this phenomenon in a total of about seven patients (12, 359, 792, 804, 811).

At times (probably more often than is indicated by the report in which it is specifically mentioned) the systolic and diastolic murmurs assume a to and fro character (12, 792). Together with a superficial scratchy quality of the murmurs, a cardiac contour approximating pericardial effusion and quiet heart borders on fluoroscopy, this feature may lead to a mistaken diagnosis of pericardial disease (429). Wood (190) (p. 333) speaks of a very characteristic superficial diastolic scratch which sounds more like a diastolic pericardial friction over the distended right atrium than a true intracardiac murmur.

#### CONGENITAL MITRAL STENOSIS

**ANATOMIC CONSIDERATIONS** Endocardial fibrosis to a certain extent associated with congenital mitral stenosis. The mitral stenosis is in such cases apparently one manifestation of the pathologic process. Congenital mitral stenosis may occur in association with other cardiovascular malformations such as coarctation of the aorta. With atrial septal defect in Lutembacher's syndrome the mitral stenosis is usually rheumatic but may be congenital. There are a few reports of a supravalvular or perivalvular variety of congenital mitral stenosis (363, 408, 1306) (p. 133 of ref. 152). The condition is often referred to as cor triatriatum for obvious reason. Most of the cases have had hemodynamic changes typical of mitral stenosis but none of the anatomic changes (1332A).

Mitral stenosis associated with congenital malformation is rare. It occurs more often in combination with other malformations. Regurgitation is much more frequently the functional result of a congenital mitral regurgitation.

**PHYSIOLOGIC CONSIDERATIONS** In terms of the pathologic physiology of the valvular lesion there is no significant difference between mitral stenosis of rheumatic and congenital origin. There may of course be associated myocardial or other valvular lesions in the case of the rheumatic disease and associated congenital malformations in the case of the congenital disorder.



matic heart disease especially when it is not accompanied by cyanosis. Reifenstein *et al* (1253) described a 20 year old Marine with Ebstein's malformation. Possibly the good prognosis should not be overemphasized, however. The long survivals cited above are in expression of a variability of the clinical picture in this malformation: sudden death occurs in this group.

In congenital tricuspid stenosis, the pathologic

physiology does not differ significantly from that in the acquired form of the disease except in connection with the fact that when congenital, tricuspid stenosis may present a less clouded picture because of the absence of lesions in other valves.

In tricuspid atresia, cyanosis always occurs because of the interatrial right to left shunt essential to life.

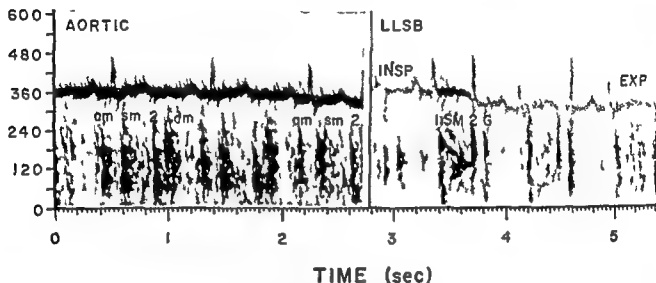


FIG. 391 Ebstein anomaly.

K. S. (763208) born in 1940 had moderate cyanosis from birth until the age of 5 or 6 years. She was, however, considered entirely normal until mid 1956 when she began having episodic precordial pain—her only complaint. The diagnosis of Ebstein's malformation was suggested by the auscultatory findings: long PR interval, low voltage of QRS on right side of precordium, incomplete right bundle branch block, prominent right atrium with mild pulsations.

The scratchy quality of the murmur is rather characteristic of Ebstein's disease. The systolic murmur is holosystolic suggesting tricuspid regurgitation.

On cardiac catheterization, the right atrium was demonstrated to be large and the tricuspid valve to be displaced well to the left of the midline. There was a double hump in the pulmonary artery pressure tracing. Femoral arteriogram (as well as the polycythemia) suggested atrial septal defect.

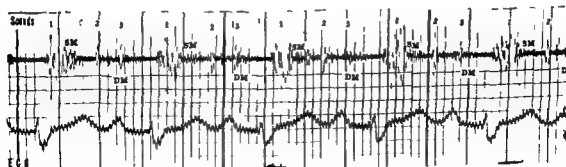


FIG. 392 Ebstein anomaly.

Prominent first sound (1), well split second sound (2), prominent third sound (3), decrescendo medium frequency moderate intensity systolic murmur (SM), mid diastolic murmur (DM) following the third sound and the P wave of the electrocardiogram (Courtesy of Ongley (1159) and *Circulation*.)

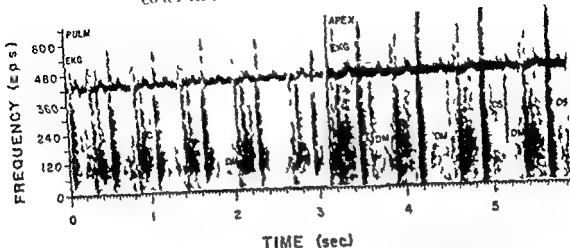


FIG. 314 Congenital mitral stenosis in R. B. (B36036) age 17 months. In the pulmonary area there is an early systolic click followed by a decrescendo murmur of ejection type. The presystolic murmur can be identified here as often the case in rheumatic mitral stenosis in young persons or any patient with a small orifice. There appears to be an opening snap. At the apex S<sub>1</sub> is snapping. The presystolic murmur is introduced by a rather clicking sound which is later than the sound interpreted as opening snap in the pulmonary area. This probably is an unusually clicking variety of the sound which often fills the opening snap and imitates the diastolic rumble in mitral stenosis.

of the aorta and diastolic rumble at the apex is often heard. For example Wood (1940 p. 355) states that he heard such a murmur in one third of cases of coarctation. Although actual congenital malformation of the valve may be present in some in the majority of these dilatation of the ventricle and relative mitral stenosis seem likely. Endocardial fibrosis to be a rather frequent association with coarctation may contribute to the production of a diastolic murmur.

In brief the auscultatory diagnosis of congenital mitral stenosis is difficult because on the one hand the characteristic diastolic murmur must be absent and on the other closely simulating signs are produced by so-called relative mitral stenosis. The presence of an opening snap strengthens the diagnosis but is not entirely pathognomonic.

Other congenital malformations of the mitral valve include cleft in association with VSD of the ostium primum type (p. 350). Lobed type malformation (78) fibroelastic change in the valve mechanism without significant obstruction. The latter may occur alone or in association with other malformations, particularly congenital aortic stenosis or coarctation (q.v.). Cleft of the anterior leaflet of the mitral valve may occur alone (408) although it is much more often associated with endocardial cushion defect. The and morbid

inversion of the chordae tendineae represent forms of congenital mitral regurgitation.

### CONGENITAL AORTIC STENOSIS

**ANATOMIC CONSIDERATIONS.** Subaortic, valvular and supra-valvular varieties of congenital aortic stenosis are described. Therefore essentially the same three varieties exist as in the case of congenital pulmonary stenosis. We have observed a similar incidence (in grandmother and grandson) of the supra-valvular variety (1140). The difficulties of differentiating congenital and rheumatic (or other acquired) basis for aortic stenosis are not only clinical but also to a considerable extent anatomical as well. The subaortic and supra-valvular type of the disease can of course be identified as congenital at least at necropsy. There is usually about one supra-valvular case for each four valvular case of congenital aortic stenosis. In the valvular type the communications between the left and non-coronary cusps is usually least affected. Early recognition of a murmur consistent with aortic stenosis is the usual clinical criterion for congenital etiology, e.g. a murmur heard before the age of four years is taken as the criterion by some. Occasionally both congenital and acquired factors are importantly involved in the causation of aortic stenosis. For example bicuspid aortic valve when it occurs

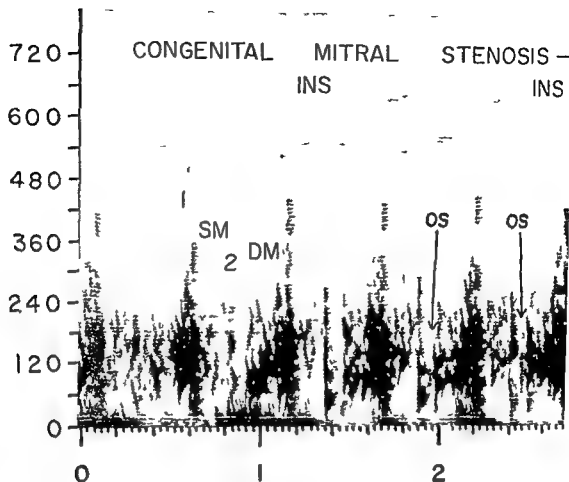


FIG. 393 Congenital mitral stenosis

Recorded at apex in C.C. (B15690) one year old child with clinical, operative and autopsy findings of mitral stenosis. Apex. Note the ringing  $M_1$ , prohibitive opening snap (OS) and typical diastolic rumble with both passive and active (presystolic) components. Compare with rheumatic mitral stenosis (e.g. Fig. 274).

**CARDIOVASCULAR SOUND** In the two well confirmed cases of isolated mitral stenosis that I have studied (Figs. 393 and 394) there has been judging by the presence of a systolic murmur at the apex and the findings at operation or autopsy some degree of mitral regurgitation. In some patients the changes otherwise have been virtually identical to those in "pure" mitral stenosis of rheumatic origin. For example take the one year old child whose murmur is portrayed in Figure 393. There is a snapping first heart sound and a diastolic murmur which begins following a short gap after the second sound and has a crescendo into the first heart sound. There may even be a faint opening snap but it is difficult to be certain because of the rapid rate. In the second case (Fig. 394) the opening snap is more definite.

Difficulties of differential diagnosis are presented by various congenital malformations which because of altered hemodynamics and/or

anatomy result in a rumbling diastolic murmur of relative mitral stenosis. Conditions of high flow across the mitral orifice—VSD, PDA—represent one group. The diastolic murmur of high mitral flow is opposed to that of structural stenosis is usually limited to mid diastole and has no pre-systolic accentuation. It often is imitated by an accentuated third heart sound. It has, therefore, the features of a Carey Coombs murmur (p. 199). The tricuspid high flow murmur in ASD may be loudest at the cardiac apex and may be distinguished with difficulty from that of mitral stenosis.

Duplication of the mitral valve that is double orifices with reduction in total orifice area has been described (333, 336, 1559) in association with mild coarctation and calcific aortic stenosis. An apical mid diastolic murmur was produced (1559).

In congenital aortic stenosis and in coarctation

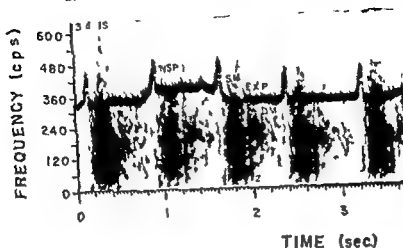


FIG. 34. Congenital aortic valvular disease.

FIG. 34. (60 sec II H - 20) 21-year-old male. He has been seen at the hospital from the age of 1 year, making the congenital nature of his lesion quite definite. Furthermore, the mother has aortic stenosis with no history of rheumatic fever and the grandmother was found at autopsy to have aortic stenosis, almost certainly congenital (Wood, 1950). In this patient (NSP1) or (EXP) the murmur could hardly have been the right most likely as often happens because of the localization of the murmur at the left sternal border. Left heart catheterization in FIG. 35 revealed a pressure of 121/36 mm Hg in the left ventricle and 83/73 (2) in the aorta. The S1C1 from FIG. 34 was shown change to a level of 45 and 48. The diastolic murmur is an unusually narrow frequency as in giving it a somewhat musical quality.

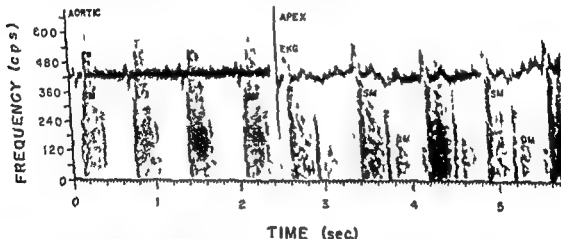


FIG. 35. (60 sec II H - 20) Congenital aortic stenosis with apical diastolic rumble.

Aortic area and apex in FIG. 35 (60 sec II H - 20) 21-year-old male. There is a typical murmur of aortic stenosis in the aortic area. At the apex there is a third heart sound following a short rumble which was quite definite 15 sec before. The rumble had not been present a year before.

ICC. As noted before, the quality of S3 is a poor index of the severity of aortic obstruction; it is also a poor index of the type of malformation which is present.

It was formerly thought that significant aortic regurgitation occurs uncommonly with congenital

aortic stenosis. It is now clear that this is not true (C. H. 196, 196).

Wood (1950) states that out of six of his patients (totaling 20) had an apical diastolic rumble suggestive of mitral stenosis, either relative or structural. The possibility of fibroelastosis

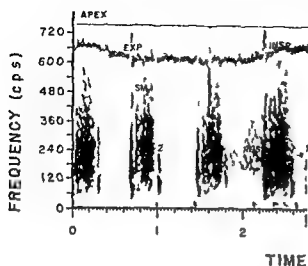


FIG 39a Congenital aortic stenosis

B C W (741719) 11 year old male had had previous evidences of left heart strain and a bout of SBI nine months previously. Right heart catheterization revealed only normal findings.

is an isolated malformation or in association with calcification of the aorta is prone to undergo atherosclerotic change (269) probably because of the disturbed flow pattern in the vicinity of the valve. Bacterial endocarditis to which bicuspid valves are prone is likely to lead to distortion of the valve. Calcification may occur in a valve so affected. Furthermore, without bacterial endocarditis, indubitably congenital aortic stenosis may show calcification of the valve and clinically show an evolution to suggest that a progressive atherosclerotic process is occurring.

Clinically congenital aortic stenosis behaves like a progressive lesion. With the passage of years the murmur becomes progressively louder and the manifestations of left ventricular strain become more pronounced. This phenomenon was well demonstrated in the following patient: W P (161996) white male had been observed at frequent intervals from the age of 17 months when he was hospitalized for a prolonged diarrheal illness. During this hospitalization many examinations made no mention of a murmur; only two recorded a soft apical systolic murmur. During the next decade the systolic murmur, which first became impressive when the patient was 4 years old, displayed a "march" from the apex to the left sternal border and finally to the aortic area (a point of maximum audibility) and rheumatic

heart disease with mitral regurgitation was entertained as the leading possibility. The electrocardiographic, radiologic, and clinical evidences of left ventricular hypertrophy and "strain" were progressive. At open heart surgery when the patient was 18 years old, a diaphragmatic subaortic stenosis was discovered.

In a case such as this it seems likely that the obstructing diaphragm does not grow (at least its orifice does not enlarge) commensurate with general and cardiac growth and with steadily increasing cardiac output. The murmur heard in earlier years at the apex, left midprecordium and left sternal border may be generated at the immediate area of obstruction. As the gradient across the aortic valve area became greater, the jet in the aorta became more pronounced and the resulting murmur in the aortic area appeared.

Experiences such as this make one hesitant to render an unequivocal judgment of benignity in the case of relatively faint murmurs in young children.

Post-stenotic dilatation of the ascending aorta is clinically demonstrable in the great majority of instances if sought by proper radiologic methods. Occasionally this phenomenon attains mammoth proportions.

PHYSIOLOGIC CONSIDERATIONS See pp 263 and 264

CARDIOVASCULAR SOUND I will speak mainly of those features which tend to be different in congenital aortic stenosis than in the familiar rheumatic or calcific aortic stenosis of adult life. Obviously none of these features is in absolute or pathognomonic differential point; otherwise there would not be the usual difficulties in differentiating congenital from acquired aortic stenosis.

Often especially in young subjects and in subaortic stenosis the murmur is loudest at the left sternal border and leads to the mistaken diagnosis of VSD.

Brofmann and Feil (184) thought that subaortic stenosis would be distinguished from aortic stenosis by the presence of a good A in the former variety. Kilo's experience (793) was not in accord with this view, however, and Mannheimer (1033) described an autopsy confirmed case of subaortic stenosis in which no aortic second sound was detected by stethoscopy or

mechanisms of the sound come to mind (1) an opening sound produced at the aortic valve (2) snapping of the wall of the aorta dilated through the phenomenon of post-tensile dilatation. The sound is probably comparable to that which occurs in milder cases of pulmonary stenosis (p. 370).

### IDIOPATHIC DILATATION OF THE PULMONARY ARTERY

**ANATOMIC AND ETIOLOGIC CONSIDERATION.** Care must be taken to exclude dilatation of the pulmonary artery secondary to ASD or mild IP. Occasionally cardiac catheterization forces one to conclude that pulmonary artery dilatation is an isolated malformation. A certain number probably small of these cases may have dilatation of the pulmonary artery as one manifestation and perhaps the main one of the Marfan syndrome (371).

Greene *et al.* (389) pointed out that it is possible to demonstrate a pressure drop across the pulmonary valve with only idiopathic dilatation of the pulmonary artery.

**CARDIOVASCULAR SOUND.** An early systolic click and a systolic murmur are to be expected. The loss of pressure energy is indicated by a pressure drop across the pulmonary valve records well with the fact that a murmur is present. Occasionally a diastolic murmur is heard and attributed to relative pulmonary insufficiency (391). The second pulmonary sound tends to be loud, probably caused mainly by improved conduction of the sound to the front of the chest possibly in part to closure of the valve from a more wide open position.

### PRIMARY PULMONARY HYPERTENSION

To what extent this condition can be considered congenital is not clear. See pages 428 to 432 for a discussion of this entity and of pulmonary hypertension in general.

**CARDIOVASCULAR SOUND.** The changes of note are (1) accentuated pulmonary second sound (2) early systolic click (3) presystolic gallop (4) pulmonary diastolic murmur.

### QUADRICEPHALIC SEMILUNAR VALVE

As discussed on p. 107 quadricephalic state is more frequently encountered in the pulmonary

valve than the aortic. Furthermore for reasons elucidated by Leonardo da Vinci about 1500 and in 1973 by Longworth (see p. 36) a four-cuspid valve is less strong in the closed position than a three-cuspid valve. Pulmonary regurgitation may occasionally develop (798). Probably this is especially likely to occur if pulmonary hypertension is present for some reason.

The early diastolic murmur of pulmonary regurgitation in this condition when uncomplicated by pulmonary hypertension may be unusually low pitched and blubbery. This statement is based on the findings in patients in whom I suspect without anatomic confirmation that the diagnosis is quadricephalic valve. Wells and co-workers (1429) have presented a loud and unusually low pitched diastolic murmur in a patient with pulmonary regurgitation. The low pressure in the pulmonary artery is consistent with a blubbery quality rather than the higher pitched more whurring quality of the Graham Steell murmur of pulmonary hypertension and the murmur of aortic regurgitation. Furthermore the murmur may have a crescendo-decrescendo—rather than pure decrescendo—pattern.

### BICUSPID SEMILUNAR VALVE

**ANATOMIC CONSIDERATIONS.** As discussed on p. 107 bicuspid condition most often affects the aortic valve. Although it may occur as an isolated anomaly it occurs frequently in association with coarctation of the aorta. Abbott (1) found it in 23.5 per cent of 200 cases of coarctation and Reizenstein, Levine and Crook (124) found it in 42.3 per cent of 104 cases. It is frequently difficult to distinguish a congenitally bicuspid valve from a valve rendered bicuspid through the rheumatic adhesion of two of its cusps (612). Particularly is this true when the atherosclerotic change to which both types of valve are prone has taken place. When there is a bicuspid pulmonary valve it is usually in association with a major malformation such as tetralogy of Fallot. However Ford and colleagues (469) described a case of isolated bicuspid pulmonary valve. Pulmonary regurgitation was present.

**CARDIOVASCULAR SOUND.** A bicuspid valve does not open completely. Therefore a systolic murmur

See references 509, 509, 510

accounting for the aortic lesion and also affecting the mitral valve to some extent has been mentioned (pp 385 and 387) Dilatation of the ventricle as at least a contributing mechanism is suggested by the experience illustrated in Figure 397 At the age of nine this patient had no apical rumble, but did have one year later In the

patient illustrated in Figure 400 a mid diastole rumble appeared at the apex in the interval of six years between two examinations

In 15 patients, Reinhold, Rudhe and Bonham Carter (1258) found a striking, proto-systolic click which was well heard at the apex where it simulated a split first heart sound At least two

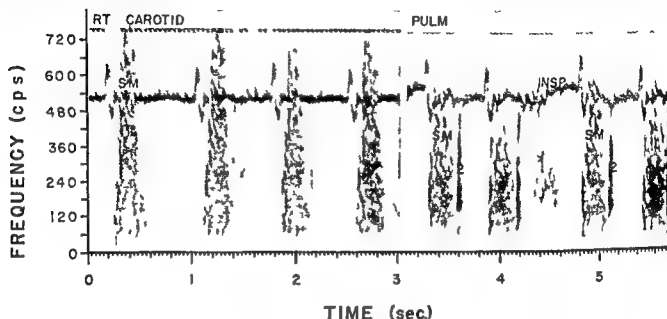


FIG 399 Congenital aortic stenosis with apical diastolic rumble

T D (733061) 3 year old male had a systolic pressure of 180 mm Hg in the left ventricle by left heart catheterization In addition to an early diastolic murmur suggesting mitral stenosis there was a pre-systolic gallop at the apex (not shown here)

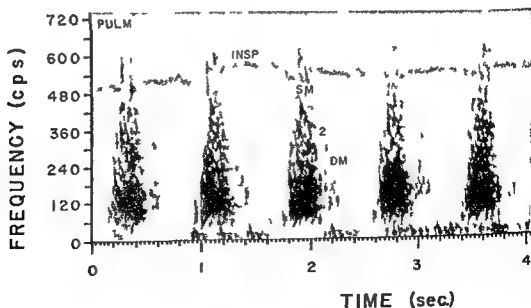


FIG 400 Congenital aortic stenosis and regurgitation are present in this 17 year old boy (S D I A90087) who has had a murmur from the first year of life Since being seen 11 years previously a mid diastolic murmur had appeared at the apex (not shown here)

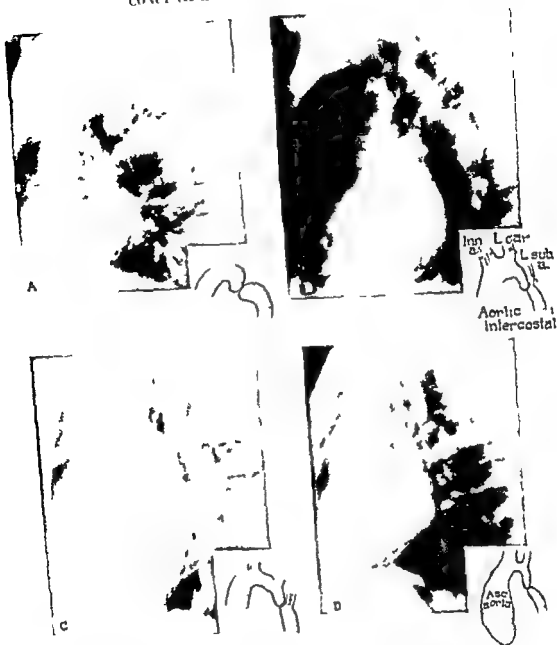


Fig. 401. Angiostriographic demonstration of the location of coarctation of the aorta in figure C. (C) dilatation of the descending aorta is demonstrated in D. (Courtesy of Cooley and Stinson (1961).)

area of the precordium e.g. at IISB. In such instances origin at the coarctation can be determined by the late onset of the murmur usually 0.12 sec or more after the first sound (see Fig. 403).

The murmur of coarctation may extend into the very first portion of the diastolic period of the ventricle merely because of the lag in transmission

of the pulse wave from the heart to the coarctated area. However a continuous murmur or a murmur with long diastolic component probably has its origin in the collaterals or in an associated patent ductus arteriosus not in the coarctated area itself. Another theoretical explanation for a murmur which extends into diastole is the principle of Myers *et al.* (1966). If mean pressure is suffi-



is to be expected. As a result of secondary change in the valve, of atherosclerotic and calcifying character, a more pronounced systolic murmur and in addition a diastolic murmur may develop. A bicuspid valve is probably not as strong in the closed position as a tricuspid, however, regurgitation is not likely to develop in the absence of hypertension and/or secondary atheromatous or bacterial change in the valve.

The systolic murmur of uncomplicated bicuspid valve is not likely to be impressive. Congenitally bicuspid aortic valve is nearly asymptomatic until complicated by atherosclerosis, bacterial endocarditis, or hypertension (1473).

### COARCTATION OF THE AORTA

(Syn. Coarctation of the aortic isthmus; stenosis of the aortic isthmus)

**ANATOMIC CONSIDERATIONS.** The most frequent site of obstruction is just distal to the ostium of the left subclavian artery in the first portion of the descending aorta (Fig. 101). This site lies in apposition to the posterior chest wall—a point of importance in connection with the radiation of the murmur. The mouth of the left subclavian artery may be narrowed or the right subclavian may arise independently of the innominate distal to the coarctation and pass to the right behind the esophagus. Collaterals providing anastomotic flow between the proximal aorta and the portion of the body beyond the coarctation are striking and account for notching of the ribs and the visible pulsations over the neck and trunk. Post stenotic dilatation occurs and is often apparent on ordinary chest x-rays as part of the 'figure of three' sign. The coarctation may be for practical purposes complete. Abbott in 1928 and Reifstein, Levine and Gross in 1947 found complete obliteration of the aorta in approximately one fourth of cases. However Edwards (580, p. 401) states that usually in opening can be demonstrated microscopically in all cases. At the other extreme the narrowing may be very slight. Take for example, the mild coarctation that frequently accompanies other more striking features of the Marfan syndrome (1071). The caliber of the aorta at the coarctation is rarely more than 2 mm. (1 D) in surgical specimens. A

jet lesion, i.e., a localized patch of atherosclerosis is sometimes seen distal to the coarctation. Progressive obliteration of the coarctated area also occurs at times. Dilatation of the ascending aorta proximal to the coarctation may occur on the basis of cystic medial necrosis, and dissecting aneurysm may develop sooner or later as a fatal complication.

The high incidence of bicuspid aortic valve (269) with coarctation is previously mentioned. Subaortic stenosis was found in 6 per cent of cases by Abbott (1). Fibroelastosis also occurs fairly frequently and may produce deformity of the mitral valve (see page 385). Mitral regurgitation and coarctation is a relatively frequent combination.

Coarctations anatomically unusual in terms of length and site in the aorta are being encountered with increased frequency (728, 737, 771, 812).

**PHYSIOLOGIC CONSIDERATIONS.** Often the flow through the coarctated area is by all evidence very small, and, of course, occasionally there is no flow at all. The characteristic feature is high pressure proximally and low pressure distally. In the distal segment of the arterial tree mean pressure although lower than that in the proximal segment may be little below normal. However, the pulse is greatly damped and the pulse pressure is small.

In experimental coarctation in dogs, Gupta and Wiggers (617) found that murmur developed when the aorta was about 60 per cent constricted, was maximal at 73 per cent constriction, and was no longer audible beyond 78 per cent constriction.

**CARDIOVASCULAR SOUND.** (1530) The same phenomena include those referable (1) to the coarctated area itself (2) to the collateral circulation (3) to the associated aortic and other valvular lesions. In addition an aortic early systolic click (ejection sound) is often heard.

The murmur produced by the coarctation itself is likely for obvious reasons to be loudest in the upper portion of the left inter-scapular area. It is usually well heard also in the left supraclavicular area and over the left upper chest anteriorly. Graphically it has, as one would predict, the configuration of an ejection stenosis murmur.

In some cases the primary murmur of coarctation is easily audible and recorded over a large

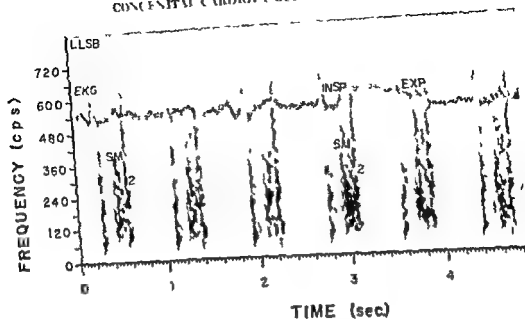


Fig 403 Coarctation

LLSB in V 4 (7/60/6) 12 year old boy. The late onset of the systolic murmur identified as the primary aortic regurgitation murmur. There was a decelerando early diastolic murmur which probably indicates aortic regurgitation. Blood pressure was 200/70 mm Hg. At the apex there was a murmur with the temporal and spectral characteristics of an opening snap.

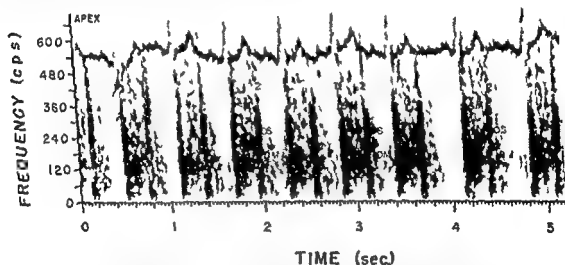


Fig 404 Mitral opening snap and apical diastolic murmur with coarctation

ApeX in V 4 (4/3/10) 12 year old boy. Marked enlargement of the left atrium was demonstrated radiologically. At operation for resection of the coarctation a systolic thrill was felt over the large tense left atrium at the site of the mural aneurysm. There was no diastolic thrill. The left atrial pressure wave suggested predominant mitral regurgitation.

efficiently may furthermore not be as strong in the closed position as a tricuspid valve. An important factor in the aortic diastolic murmur in many cases is almost certainly dilatation of the

base of the aorta with stretching of the ring set at the aortic cup. Cystic medial necrosis may be the anatomic substrate of this dilatation in some instances (644). Aneurysm of the sinus of Valsalva

ciently lower distal to the coarctation than proximal, the murmur may extend into diastole and even a continuous murmur might be produced, merely because pressure proximal to the coarctation is higher during all parts of the cardiac cycle. The view that extension of the murmur into diastole is due to backflow during that phase is untenable. The primary murmur of coarctation is absent when the coarctation is complete and disappears with progressive obliteration. Wood (1900) found no inter-scapular murmur in about 15 per cent of cases. When the coarctation is at an unusual site the murmur may be heard in an anomalous position. For example in cases of abdominal coarctation, the murmur was in the epigastric and upper lumbar regions (272, 737, 728, 771-812).

A continuous humming murmur with systolic accentuation arising in the collaterals may be present at various sites over the upper back. At times this murmur may be only systolic with its onset possibly later in systole than the murmur generated at the coarcted area. Occasionally it is possible to relate the murmur to a specific superficially located anastomotic vessel; the murmur may be obliterated by pressure on the vessel (272). Sometimes the murmur in the back in the area of the coarctation is probably produced by blood entering the distal aorta in a collateral because the coarctation may be essentially complete (272). On the basis of a few patients who,

albeit young and therefore subject to venous hums anyway, have had very striking murmurs of this character in the neck (Fig. 199), I have come to view venous hums as a frequent feature of coarctation.

An "aortic" systolic murmur occurs in the majority of cases of coarctation. Causes operative alone or in combination are (1) transmission of the primary coarctation murmur or murmurs of collaterals, (2) bicuspid aortic valve, (3) aortic stenosis (Fig. 402), (4) subaortic stenosis, (5) dilatation of the aorta. It is often exceedingly difficult in a given case to be certain which factor(s) is responsible (272). Campbell and Bixler (22a) placed the incidence of aortic stenosis at 0 per cent.

An aortic diastolic murmur occurs in a significant proportion of cases of coarctation. In one series totaling 130 patients (225) such a murmur was present in almost one third. It was present in 29 of 124 cases, excluding 6 with aortic stenosis (22a). Wood (1900 p. 338) found it in 10 per cent. In 96 cases at the Mayo Clinic it was present in 20 per cent. That the hypertension is at least indirectly responsible seems likely. Secondary atherosclerotic change in a bicuspid valve may, with the hypertension, account for the diastolic murmur in most cases; the incidence and severity of aortic regurgitation increases with age (22a). A simple bicuspid valve which does not open

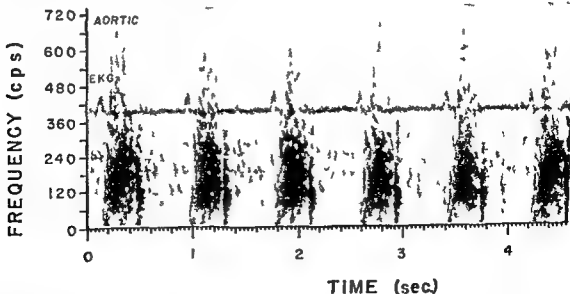


FIG. 402 Aortic stenosis as coarctation. Suprasternal notch in I W (762010) 53 year old male with coarctation. The murmur is typical of aortic stenosis. It occurs too early to be the murmur of the coarctation itself. (Much background noise is present.)

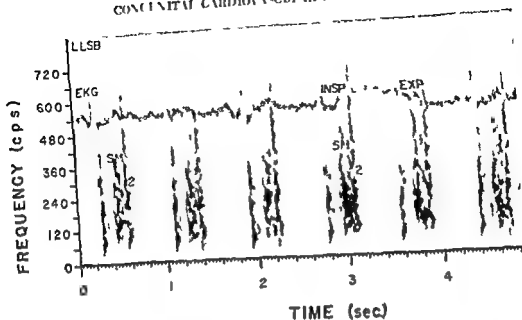


FIG 403 Coarctation

LLSB in W (740310) 6 year old boy. The late onset of the systolic murmur identifies it as the primary coarctation murmur. There was a decrease in early diastolic murmur which probably indicates aortic regurgitation. Blood pressure was 95/70 mm Hg. At the apex there was a sound with the temporal and spectral characteristics of an opening snap.

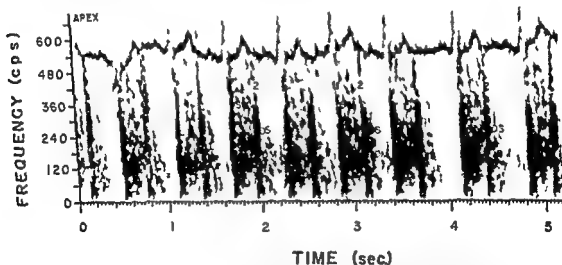


FIG 404 Mitral opening snap and aortic diastolic murmur with coarctation

Apex in H. B. (740310) 6 year old boy. Marked enlargement of the left atrium was demonstrated radiologically at operation for resection of the coarctation as a thrill was felt over the large tense left atrium at the site of the mural leaflet. There was no diastolic thrill. The left atrial pressure wave suggested predominant mitral regurgitation.

efficiently may furthermore not be as strong in the closed position as a tricuspid valve. An important factor in the aortic diastolic murmur in many cases is almost certainly dilatation of the

base of the aorta with stretching of the ring, so that of the aortic cusps. The medial aortic arch may be the anatomic substrate of this dilatation in some instances (644). Aneurysm of the sinus of Valsalva

has been demonstrated in association with coarctation (374), and a diastolic aortic murmur is likely to be present in such cases (360)

An important point is that when the aortic regurgitation associated with coarctation is of severe degree, as it sometimes is, the signs of coarctation may be submerged (for the unwary). The femoral pulses may feel quite adequate, if it is not appreciated that the water hammer pulse present in the arms is not felt there and that there is, in fact, a delay in the femoral pulse.

Wood (1590, p. 335) states that one third of his patients with coarctation had an apical diastolic murmur suggesting mitral stenosis. In this enumeration he excluded five patients with associated ASD or PDA which alone may be accompanied by such a murmur and also excluded three patients with rheumatic heart disease and mitral stenosis. Cleland and his colleagues (272) found the same apical mid diastolic murmur without other evidences of mitral stenosis in 15 of 40 operated cases. They were impressed with its short duration, less rumbling quality, and its constant nature. Of the 15 patients two had persistent ductus arteriosus and six had in early decrescendo diastolic murmur at the left sternal

border. Size of the left ventricle could not be related to presence or absence of the murmur in any convincing manner. In six of the patients the mitral murmur was not heard during post operative observations. However, this notation is of doubtful significance because of the inconstancy of the murmur before operation. We have noted (see Figs. 401, 405, 406) the occurrence of what appears to be an opening snap in at least two patients with coarctation (P. B., 775818, K. B. 740310). The coarctation was of the type in these patients, the left atrium was enlarged in the first, however.

The same explanations for the diastolic murmur (Figs. 404 and 407) are applicable as in the case of the same murmur occurring with congenital aortic stenosis, dilatation of the ventricle with relative stenosis or fibroclastic change (1162) in the mitral valve with actual obstruction. Actual mitral stenosis has been described with coarctation as an associated congenital malformation (741, 1156).

Because of the hypertension and the frequent presence of dilatation of the ascending aorta, it is not surprising that in early systolic click is often heard in the aortic area.

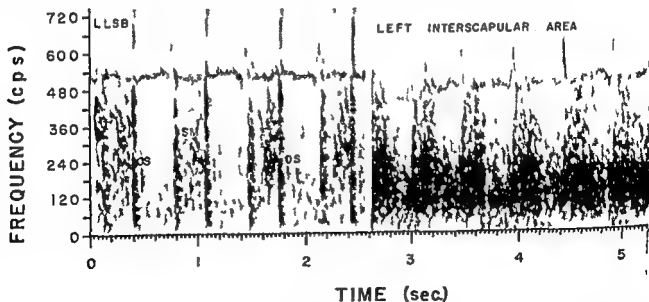


Fig. 405 and 406 Mitral opening snap with coarctation

LLSB and interscapular area of back in I. II (775818) 18 year old girl. A murmur was present from birth. Pulmonary capillary pressure was 14 mm Hg. The left atrium was enlarged by x-ray. A diastolic murmur at the apex showed a presystolic decrescendo. In some other areas the abrupt and late onset of the systolic murmur is a feature characteristic of coarctation which is more clearly demonstrated. The nondecript continuous low frequency noise is thought to be produced in arterial collaterals (left interscapular area).

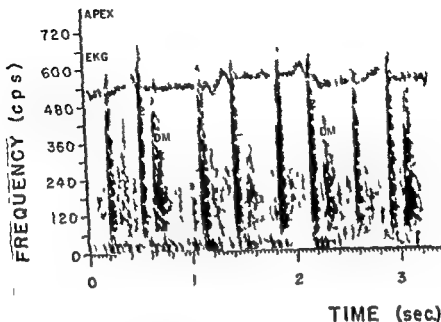


Fig. 40. Coarctation with congenital mitral stenosis.

Apex in R T (30 60s) who in addition to the apical diastolic rumble has (1) angiocardiography enlargement of the left atrium and delay of the dye in the chamber

After operation there may be slight residual coarctation as indicated by a brachial artery pressure higher than femoral (1993). A systolic murmur may remain in such cases.

**PSEUDO COARCTATION.** There is a condition variously known as kinked aorta (188, 194) buckled aorta atypical coarctation (1280) subclinical coarctation (1417) pseudo-coarctation (1437) in which there is distortion and often mild narrowing of the aortic arch at the level of the insertion of the ligamentum arteriosum. On ordinary x-ray of the chest the anomaly may simulate a tumor in that area. The anomaly may occur in the Marfan syndrome and may be associated with aneurysm of an aortic sinus of Valvula. In patients with or without the Marfan syndrome Bruner and Burchell (188) found a systolic murmur at the base of the heart in 9 of 10 cases and attributed it to turbulence of blood in the vicinity of the aortic kink. In two of the 9 patients the murmur had been detected in childhood and had been the basis for a diagnosis of organic heart disease. DiGuglielmo and Cuttaduro (364) reported a similar experience. The clinical observation seems consistent with those of Cup and Wigener (617) who found that about

60 per cent constriction was necessary for murmur production in experimental coarctation.

#### CONGENITAL PULMONARY REGURGITATION

**ANATOMIC CONSIDERATIONS.** Isolated pulmonary regurgitation is an exceedingly rare congenital lesion. At times the anatomic substrate is a quadrifid valve as mentioned previously. In several of the clinical reports the anatomic basis for regurgitation has not been known. It is difficult clinically to differentiate isolated pulmonary regurgitation on other also rare bases such as healed gonococcal endocarditis (1152).

Pulmonary regurgitation is occasionally found in association with other congenital lesions such as Lillienberg's syndrome (in the case of LaVenne *et al* (835) there was total absence of pulmonary cup) pulmonary stenosis and ASD etc. Camperio *et al* (229) have also reported a case of total absence of pulmonary valve with ASD. A remarkable feature was survival to the age of 32 years and performance of heavy work. Death was due to coronary occlusion.

**PHYSIOLOGIC CONSIDERATIONS.** On fluoroscopy there is likely to be striking hilar distention and by electrokymograms or roentgenkymograms large

has been demonstrated in association with coarctation (374), and a diastolic aortic murmur is likely to be present in such cases (360).

An important point is that when the aortic regurgitation associated with coarctation is of severe degree, as it sometimes is, the signs of coarctation may be submargined (for the unwary). The femoral pulses may feel quite adequate if it is not appreciated that the water hammer pulse present in the arms is not felt there and that there is in fact a delay in the femoral pulse.

Wood (1990, p. 335) states that one third of his patients with coarctation had an apical diastolic murmur suggesting mitral stenosis. In this enumeration he excluded five patients with associated VSD or PDA which alone may be accompanied by such a murmur and also excluded three patients with rheumatic heart disease and mitral stenosis. Cleland and his colleagues (272) found the same apical mid diastolic murmur without other evidences of mitral stenosis in 13 of 40 operated cases. They were impressed with its short duration, less rumbling quality and inconstant nature. Of the 13 patients two had persistent ductus arteriosus and six had in early decrescendo diastolic murmur at the left sternal

border. Size of the left ventricle could not be related to presence or absence of the murmur in any convincing manner. In six of the patients the mitral murmur was not heard during post operative observations. However, this notation is of doubtful significance because of the monotony of the murmur before operation. We have noted (see Figs. 404, 405, 406) the occurrence of what appears to be an opening snap in at least two patients with coarctation (P.B., 770818, K.B. 740310). The coarctation was of classic type in these patients, the left atrium was enlarged in the first, however.

The same explanations for the diastolic murmur (Figs. 404 and 407) are applicable as in the case of the same murmur occurring with congenital aortic stenosis, dilatation of the ventricle with relative stenosis or fibroelastotic change (1162) in the mitral valve with actual obstruction. Actual mitral stenosis has been described with coarctation as an associated congenital malformation (741, 1136).

Because of the hypertension and the frequent presence of dilatation of the ascending aorta it is not surprising that an early systolic click is often heard in the aortic area.

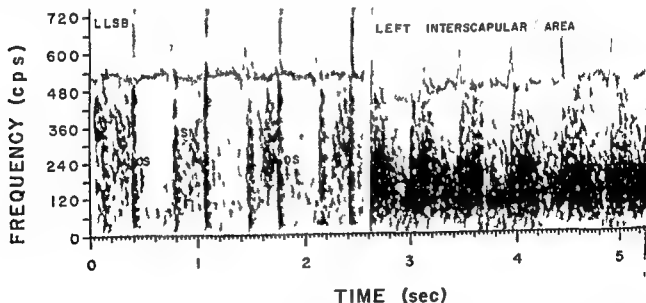


FIG. 405 and 406 Mitral opening snap with coarctation

LLSB and interscapular area of beat in P.B. (770818) 18 year old girl. A murmur was present from birth. Pulmonary capillary pressure was 18 mm Hg. The left atrium was enlarged by x-ray. A diastolic murmur at the apex showed a presystolic crescendo. In some other areas the abrupt and late onset of the systolic murmur, a feature characteristic of coarctation, was more clearly demonstrated. The nondescript continuous low frequency noise is thought to be produced in arterial collaterals (left interscapular area).

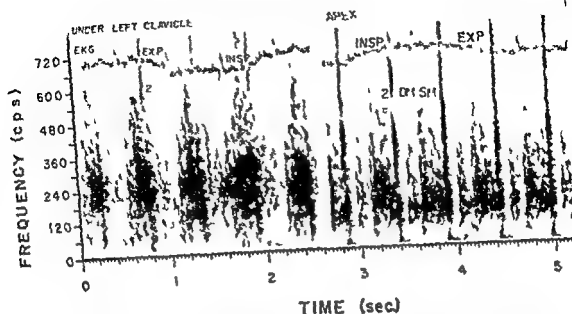


FIG. 410 Typical PDA

Left subclavicular area (left) and apex (right) in D.K. (61.83) 5½ year-old female. The peak of intensity and frequency in the continuous murmur precedes  $\frac{1}{2}$  by a significant interval. There is a mild aortic murmur at the apex.

children particularly infant there is normally less aorto-pulmonary pressure differential. What difference in pressure exists may be greater in many other children and colleagues (621) demonstrated no diastolic gradient in a patient with PDA and pulmonary hypertension who demonstrated only a systolic murmur. Pudolph and Mixer (1319) had a similar experience in a group of 20 infants (one year old or less) without continuous murmur. The aorto-pulmonary artery pressure gradient is closely related to murmur production in PDA.

When a PDA occurs proximal to coarctation of the aorta the shunt from aorta to pulmonary artery is exaggerated. When PDA occurs distal to a coarctation or when pulmonary hypertension is present the direction of shunt is likely to be reversed at least during a systolic characteristic of the so-called reversed ductus is evident in the feet and sometimes in the left arm without cyanosis in the other parts.

Patency of the ductus arteriosus can be demonstrated to persist for as long as fifteen hours after birth in the human by a dye method (1232) by differential arm leg oxygen saturations (40) and by cineangiographic observation (930). Comparable demonstrations by use of the murmur

have been reported by Daves and colleagues (1733, 339) in newborn lambs in which a typical murmur of patent ductus is almost invariable. Why the murmur is not likewise the rule in newborn humans is discussed without definite conclusion by Born and his co-authors (177).

Physiologic factors which influence aorto-pulmonary pressure differential are likely to influence the volume of the shunt and the intensity of the murmur. Pulmonary hypertension has already been mentioned. Shepherd *et al* (1361) were able to reverse a right-to-left shunt in a patient with pulmonary hypertension by having the patient breathe 99.6 per cent oxygen, a procedure known to reduce pulmonary arterial pressure.

It is interesting to reflect on the fact that with PDA the left ventricle labors under a diastolic overload whereas the right ventricle may, if there is pulmonary hypertension be exposed to a systolic overload (1702). The diastolic overload is responsible for the fact that left ventricular ejection is prolonged relative to that of the right ventricle (621) and in turn for the paradoxical splitting of S (587).

Bacterial endocarditis is one of the major risks



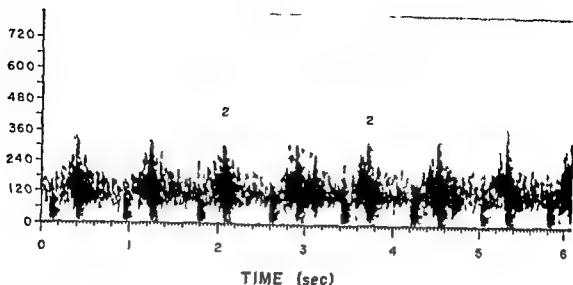


FIG 408 Patent ductus arteriosus

Typical continuous Gibson murmur with peak intensity and frequency in the vicinity of  $S_1$ —actually slightly before  $S_1$  Pulmonary area

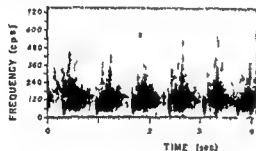


FIG 409 Patent ductus arteriosus

Typical Gibson murmur Pulmonary area

excursions of border movement in the main pulmonary artery with rapid rise and fall. By endre catheterization there is usually a wide pulmonary arterial pulse pressure. Particularly diagnostic is the presence of a sharp slope of the ejection limb with fall in pressure in diastole to a level almost equal to that in the right ventricle (1117). Using these features the clinical diagnosis of isolated pulmonary regurgitation was made by Kjellberg *et al* (800), Lord (469) and Morton and Stern (1117).

The clinical benignity of pulmonary regurgitation even absence of the pulmonary valve is consonant with the findings in animals with experimentally produced lesions of the pulmonary valve. One cannot produce heart failure in dogs with only avulsion of the pulmonary cusps (52). It is likely that because of the low level of pressure in the pulmonary artery there is relatively little regurgitation.

**CARDIOVASCULAR SOUND** The only feature of note is the early diastolic murmur which, in the absence of pulmonary hypertension, is likely to be low pitched and on the whole not conspicuous. Holldick and Wolf (706) have noted a gap between  $S_1$  and the onset of the murmur in pulmonary regurgitation. Sometimes the murmur is *crescendo decrescendo* rather than strictly *decrescendo* from the second sound.

#### PATENT DUCTUS ARTERIOSUS (PDA)

(Syn. Patent ductus botalli; patent arterial duct)

**ANATOMIC CONSIDERATIONS** The usual site of the communication is between the aorta in the latter (or posterior) portion of the arch opposite the left subclavian ostium and the first part of the left main pulmonary artery. A jet lesion may occur in the intima of the pulmonary artery opposite the opening of the ductus. Occasionally, in association with coarctation, the PDA communicates with the aorta distal to the coarctation. Associated aortic or subaortic stenosis has been emphasized recently by Mink and colleagues (1041).

**PHYSIOLOGIC CONSIDERATIONS** Because the pressure in the aorta is normally higher than that in the pulmonary artery at all times in the cardiac cycle the flow is at all times from aorta to pulmonary artery in uncomplicated cases of PDA. The normal pressure gradient between the aorta and pulmonary artery is charted in Figure 101. In

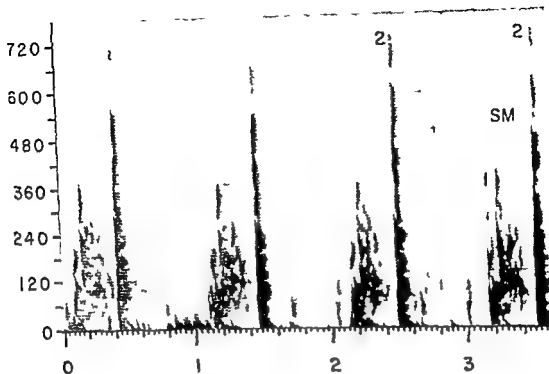


FIG. 419 Patent ductus arteriosus with pulmonary hypertension

1 pulmonary area 1 early systolic click initiates decreasing systolic murmur 1 greatly accentuated S<sub>2</sub> C<sub>1</sub> on murmur

when the diagnosis of PDA was made by aortography. Keith and Forst (776) found a typical continuous murmur in three in two early systolic murmur which at times extended into early diastole in two no murmur at all and finally in two early systolic murmur only located in the pulmonary area.

Hjellberg *et al* (800) have pictured a continuous murmur which was recorded in an infant ten days old but which had disappeared completely by the time the child was four months old. A similar experience in two other patients was recorded. Presumably delayed but adequate closure of the ductus had occurred.

Padolph and Moyer (1319) described 22 infants aged 1 to 12 months, only two of whom had a continuous murmur. The absence of the Gibson murmur could not be attributed to lack of lung since in 20 pulmonary flow was about twice systemic flow. There was however no aorto-pulmonary pressure gradient during diastole. All the infants showed poor growth and development. Difference in the murmur in various cries may be the result of elevation of different types of patients for study.

If the Gibson murmur is not quite continuous it is sometimes possible to demonstrate a gap of about 0.06 sec between the first sound and the onset of the murmur (1210).

Sometimes in children the continuous murmur is replaced by only a systolic murmur during attacks of some condition producing partial or pharynx (800). With the advent of left ventricular failure or the elevation of pulmonary artery pressure due to bronchopneumonia the continuous murmur is likely to disappear (227, 332) presumably because of elevation of pulmonary arterial pressure. I have known of obliteration of a PDA and disappearance of the characteristic murmur probably as a result of thrombosis accompanying bacterial endarteritis which was also cured spontaneously. Bishop (107) described a case in which closure was thought to have occurred between seven and fourteen years, also possibly from endarteritis. Spontaneous cure of bacterial endarteritis (even without closure of the ductus) was described before the days of chemotherapy (237). Jager (712) observed obliteration from thrombosis occurring on marked rheumatism in a 13-year-old woman. In the case of Chiles and Colquhoun (260) a sponta-

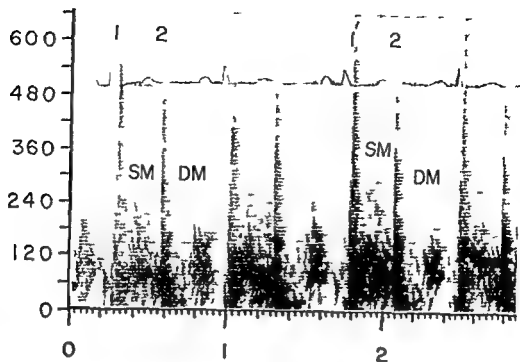


FIG. 411 Patent ductus arteriosus  
Mid diastolic rumble at the apex

in patent ductus arteriosus. It occurred in 23 per cent of Abbott's cases (2) and in 28.6 per cent of the cases of Gelfman and Levine (538). Spontaneous closure of the ductus is a result of the thrombosis induced by bacterial infection has been reported (see below).

**CARDIOVASCULAR SOUND.** The characteristic murmur called the Gibson murmur for George A. Gibson (546) of Edinburgh (see p. 29), is a continuous machinery murmur which has maximal intensity and frequency span in the vicinity of the second heart sound. The murmur seems to be built around the second sound which it envelopes (638, 1316). It is usually loudest in the first and second left intercostal spaces under the left clavicle.

The reason for the shape of the Gibson murmur is not immediately evident since the peak of aorto-pulmonary pressure differential (Fig. 100) occurs earlier than the second heart sound where the peak of the murmur is usually situated. As the pressure differential increases the size of the ductus may increase and there may be a resulting phase shift in the murmur. Münchheimer (1033) states that the larger the ductus the earlier is the peak of the murmur. In general close scrutiny of the Gibson murmur usually indicates that its peak is late systolic and not exactly coincident with S

Ziegler (1603) scrutinized the two following views which were held rather generally prior to his study:

1 The usual time of appearance of the typical murmur is generally stated to be at the age of three to five years.

2 Below this age the continuous murmur is supposedly infrequent and its presence justifies the suspicion of either the presence of a complicating defect such as pulmonary stenosis or coarctation of the aorta or an entirely different diagnosis than patent ductus arteriosus.

In a series of 20 cases in individuals of an age three years or less Ziegler's findings (1603) were as follows:

1 Except in the presence of a complicating pulmonary hypertension a typical continuous murmur was invariably present by the age of 18 months.

2 A continuous murmur may be and frequently was present in infants with an uncomplicated patent ductus arteriosus as illustrated by two patients aged six weeks and five months respectively.

In an addendum Ziegler referred to an infant five and one half and another eight months old each with continuous murmur. Adler (9) heard a continuous murmur as early as six weeks. Holliday and Wolf (706, p. 128) present the phonocardiogram of an infant with PDA and a continuous murmur in the first days of life. In nine infants in

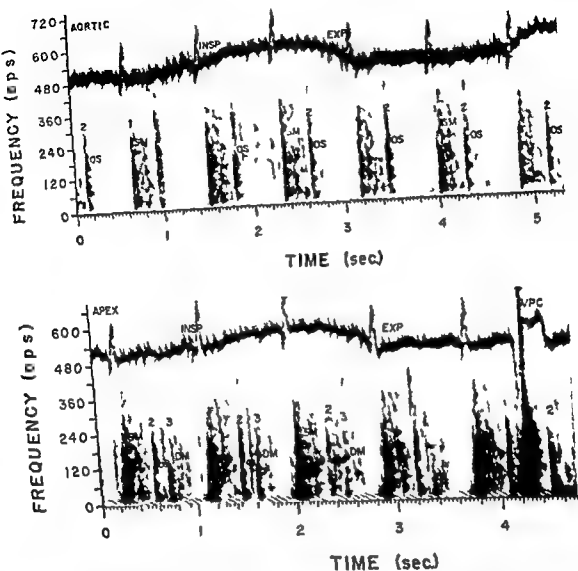


FIG. 414 Opening snap and diastolic rumble in I T (333) a 70 year old man with wide patent ductus arteriosus. Aortic (above) and apical (below) areas. Both snap and rumble disappeared after ligation of PDA.

aortic right-to-left shunt (Fig. 412). There may be a diastolic murmur of pulmonary regurgitation (see Table 16). Most reports (790-1000, 1384-1481) of PDA in adults without typical murmur principally concern cases with pulmonary hypertension (1133). Holldrick and colleagues (702) described a 16-year-old girl with a large ductus and essentially identical aortic and pulmonary pressures. The only murmur was diastolic and was probably produced by pulmonary regurgitation. The murmur was rough non-decrescendo and widely transmitted especially toward the left

shoulder. I have experienced others confused it for a systolic murmur.

Only rarely does a typical continuous murmur once established disappear. When it does disappear as in a few reported cases (8, 186) spontaneous closure is one poorly documented possibility (see p. 401) and the development of pulmonary hypertension is another. It is probable that most cases of PDA with pulmonary hypertension are that from birth. Obviously the wide PDA (see below) likewise that from birth.

A PDA entering the aorta at or beyond a

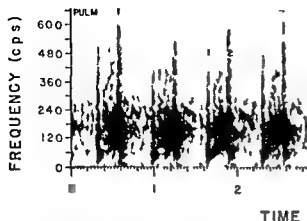


FIG. 413. Murmurs in murmur of PDA

S.H. (352841) 18 years old shows a continuous murmur typical of PDA except for the evidence of murmur in diastole

taneous closure was discovered to have occurred 15 years after the onset of subacute bacterial endocarditis. The infection had been treated with a sulfonamide in several courses.

Soulié and his colleagues (1418) by means of a balloon catheter introduced into the aorta by the usual method of right heart catheterization, were able to occlude a ductus arteriosus and abolish the continuous murmur. Failure of the murmur to disappear was proposed as a test for associated cardiovascular malformation.

There is often in the pulmonary area an early systolic click related to the dilated pulmonary artery.

At the apex there is likely to be a mid diastolic Carey Coombs type of murmur when the volume of the aortic pulmonary shunt is large (Figs. 410, 411 and 414). Ray and Darley (1248) found such a murmur in 9 of 21 patients. Actual mitral stenosis due to fetal fibroelastosis may occur with patent ductus arteriosus (38, 145, 224, 682, 1162). The differentiation of functional from organic mitral stenosis may be difficult. Left atrial enlargement may occur in both. In organic stenosis an opening snap and a presystolic element of the murmur at slow heart rates are more likely to occur and pulmonary capillary pressure at right heart catheterization is likely to be higher.

Paradoxical splitting of  $S_2$  was found by Gray (587) in 10 of 29 cases of PDA: the aortic component followed the pulmonary component and splitting was greatest in expiration. This splitting is thought to be related to the discrepant stroke

volumes of the ventricles, that of the left ventricle being larger. Systole in the left ventricle is prolonged in relation to the right ventricle. Splitting is not present more often because of the partial cancelling effect of the normal aortic pulmonary closure sequence.

With a large patent ductus arteriosus there may be Durozier's sign. In Routier's (1419), the sign was present in eight. Other peripheral signs usually associated with AR may also be present.

**ATYPICAL PDA.** In the "reversed ductus" syndrome—patent ductus arteriosus with pulmonary hypertension and reversed shunt—there may be no murmur or only a systolic murmur related either to the dilated pulmonary artery or to the

TABLE 16

Murmurs in selected patients with balanced or reversed PDA proved by oxygen saturation studies or other studies\*

| Author                       | Patient No. | Age (years) | Murmurs  |
|------------------------------|-------------|-------------|--|
| Cochran (1953)               | 1           | 27          | Faint pulmonary systolic                           |
|                              | 2           | 9           | Loud systolic over precordium                      |
| Hultgren <i>et al</i> (1953) | 2           | 31          | No murmur  |
|                              | 1           | 31          | Systolic ILSB                                      |
|                              | 4           | 21          | Systolic ILSB                                      |
| Dimmick <i>et al</i> (1953)  | 2           | 29          | Pulmonary systolic and thrill diastolic at ILSB    |
|                              | 3           | 20          | Systolic ILSB                                      |
|                              | 4           | 30          | Pulmonary systolic and diastolic                   |
| Smith (1951)                 | —           | 19          | Systolic third left inter space                    |
| Emalie Smith (1955)          | —           | 10          | Loud systolic maximal in pulmonary area            |
| Whitaker <i>et al</i> (1955) | 2           | 26          | No murmur  |
|                              | 3           | 41          | Pulmonary systolic                                 |
|                              | 4           | 53          | No murmur  |
|                              | 5           | 22          | Pulmonary diastolic                                |
|                              | 6           | 46          | No murmur  |
|                              | 7           | 8           | Pulmonary systolic and diastolic                   |
|                              | 8           | 34          | Pulmonary systolic                                 |
| Campbell (1955)              | 28          | 19          | Pulmonary systolic                                 |
|                              | 29          | 19          | Rough pulmonary systolic faint pulmonary diastolic |
|                              | 30          | 40          | Loud pulmonary diastolic                           |
|                              | 32          | 27          | Soft pulmonary systolic                            |

\* Adapted from Born *et al* (1977) with additions

### CONDITIONS WITH AUSCULTATORY SIMULATION OF IDA

Probably a generalization of the statement made by Muir and Brown (1131) in 1933 is true that in its typical form the Cibaon murmur is the most pathognomonic of all murmurs. Yet as experience increases the list of simulating conditions is enlarged.

Any intrathoracic arteriovenous fistula (PDA is essentially such) may be accompanied by a machinery murmur of the Cibaon type (316) (Fig. 416-417). Examples are aortic valvular defect or aorto-pulmonary window (see later) and rupture of an aneurysm of the aorta of the descending pulmonary artery, right ventricle or right atrium. Also the artificial ducts created by the Blalock-Taussig and Lott operation produce a murmur of this character.

Fistulous communication between a coronary artery and the coronary vein (1189) or sinus (331), a chamber of the heart (762) or the pulmonary artery (103) may be accompanied by a Cibaon murmur (Fig. 418). Sometimes the murmur is predominantly diastolic or has a diastolic not a late systolic accentuation caused by the fact that coronary flow is maximal during diastole. Knobloch and Rawson (806) reported the case of a 33-year-old business man whose murmur discovered at age 18 was described as a peculiar harsh Crude III diastolic apical murmur of blowing character. The location of the murmur will help differentiate coronary AV fistula from IDA in those cases in which it is maximal over the lower precordium or apex. However cases occur in which the murmur is located high on the left (1004). David and colleagues (331) reported a 38-year-old patient in whom a huge communication between the left circumflex coronary artery and the coronary sinus resulted in a loud continuous murmur in the pulmonary artery wide pulse pressure and high output cardiac failure. The superficial quality of the murmur may suggest that coronary AV fistula is responsible. (The oxygen content of coronary sinus blood ordinarily very low is likely to be high in cases of coronary AV fistula). However if the communication is with the right ventricle or pulmonary artery the

oxygen step up at cardiac catheterization will be found at these sites. Recently Edwards (110A) and others (211B) have resurrected the old observation (181A) that flow in an anomalous right coronary artery in an aneurysm of the pulmonary artery is toward the pulmonary artery. The development of an arterio-arterial fistula which functionally is an arteriovenous fistula may occur.

The AV fistula may be in the thoracic cage—internal mammary (103A, 103) or intercostal—and be either congenital or the result of rib fracture or rib wound.

Arteriovenous fistulas which are fundamentally congenital may not enlarge to the point that clinical manifestations are produced until rather late in life (593). Congenital AV fistulas are in many instances progressive not static lesions. The progressive character is noted in connection with AV fistulas in the extremities in the lungs and at most other sites.

In many but not all cases of pulmonary arteriovenous fistulas a murmur may be audible in overlying area of the chest (1033). It is likely to be a continuous murmur but may be loud in inspiration and almost inaudible with expiration (1127). Other writers (706 p. 112) describe the murmur as being, but loud in inspiration. Judging by the reports in the literature one (1101) can conclude that about two third of patients show a murmur directly attributable to the fistula(s). The hant of blood is occasionally of sufficient volume to result in a functional mitral diastolic murmur. The characteristic syndrome includes cyanosis, polycythemia and clubbing of the fingers. Heart failure does not occur commonly contrary to the situation with peripheral AV fistula, probably because a fistula large enough to cause heart failure would kill sooner by its effect on arterial oxygen saturation. The patient may get into one or more of three main types of serious trouble: (1) hemoptysis which may be fatal; (2) infection that is bacterial endocarditis on the fistula; (3) neurologic complications—thromboses secondary to the polycythemia and brain abscess which occurs with increased incidence in this as in other varieties of right to left shunt. The fact that about one half of all cases of the pulmonary AV fistula occur is part of the Oller Renda Weber syndrome (1021) is indicated.

See reference 333 and 1110.

correlation is likely to be a "reverse ductus". The auscultatory findings are atypical and do not permit diagnosis by this means alone.

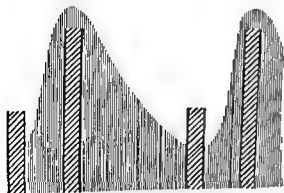
The second major category of atypical PDA is what Aitken in his excellent study (14) refers to as "the wide patent ductus arteriosus". There may be moderate pulmonary hypertension, but what hypertension is present is more the effect of high flow through the pulmonary circuit: the volume of pulmonary flow in these cases may be at least three times that of systemic flow. The clinical features Aitken emphasizes are (1) the presence of a suprasternal thrill which often is also present in the neck vessels, and (2) the replacement of the typical Gibson murmur by a systolic diastolic murmur resembling that of aortic stenosis and regurgitation. The patients usually demonstrate left atrial enlargement and in typical diastolic rumble. Even a mitral opening snap may occur in these cases in the absence of organic change in the valve (Fig. 414). Neill and Mounsey (110A) had five patients with PDA with opening snap. In all but one the snap disappeared after operation. Elevation of pulmonary capillary pressure (to an average of 19.6 mm Hg in 14 patients) agreed well with these other signs of relative

mitral stenosis. Collapsing peripheral pulse and wide pulse pressure was more likely to be present. Early severe disability is usual. Mannheim and Sandblom (1036) and Dammann and Sell (328) together have described 21 cases of wide ductus and also emphasize that the murmur is likely to be systolic diastolic or only systolic. The thrill in the neck vessels and suprasternal notch is probably produced as a result of the high flow through the aorta. Pertinent to the absence of the typical Gibson murmur may be the characteristic pressures recorded at cardiac catheterization. In the region of the pulmonary artery near the orifice of the ductus the high pressure of the aorta is transmitted to the catheter whereas both proximal and distal to that point pressures are lower. Several authors (1486) have emphasized the 'mitral syndrome' or 'pseudo mitral syndrome' which tends to be unusually striking in the patient with a large ductus: typical rumble, enlarged left atrium, left atrial hypertrophy by electrocardiogram, elevated pulmonary capillary pressure, gradient across the mitral valve demonstrated it operation.

Wide PDA can be difficult to differentiate from aortic septal defect.

### THORACIC A-V FISTULA

(e.g. patent ductus arteriosus, thyroid bruit, Blalock-Taussig anastomosis, pulmonary, coronary and other intrathoracic A-V fistulae)



### AORTIC STENOSIS AND REGURGITATION

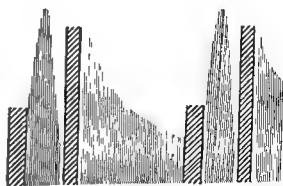


FIG. 415 Comparison of the frequency-time pattern of the murmur of thoracic arteriovenous fistula with that of aortic stenosis and regurgitation. (The Christmas tree murmur of aortic stenosis should have been indicated as stopping with a brief gap before S<sub>2</sub>.)



FIG. 419A and 419B Suspected arteriovenous fistula of the coronary circulation

A continuous murmur was audible over the lower sternum at the level of the fifth intercostal space. The patient, a 10-year-old boy, had no symptoms (Fig. 419). Anteroposterior and (Fig. 419) lateral view, exposure 1.6 sec. after the injection of contrast medium. At the time of aortic opacification a dilated tortuous aorta (arrows) was filled (Courtesy of Cooley and Sloan (1961)).

by family history or the presence of telangiectases of the skin and mucous membrane can be used in diagnosis. Telangiectases should be sought on the lip and elsewhere and the family examined.

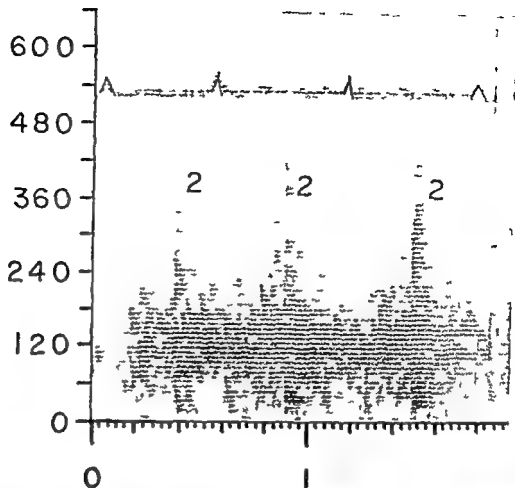
Claiborne and Hopkins (266) describe a case with PDA-like murmur in which an AV fistula in the lung represented a communication between a branch of the aorta and the pulmonary vein, rather than pulmonary artery and pulmonary vein. See Fig. 214 for a similar case of mine.

With defect of the aorto-pulmonary septum (289, 333, 332, 320, 349, 330, 613, 811) the peripheral signs of PDA, such as wide pulse pressure and usually are present. The murmur is usually said to be situated lower than that of PDA, being maximal right over the sternum at the level of the second and third intercostal spaces or at the left sternal border in the axilla. However this differentiating point has been questioned (39). The murmur has been continuous in a few patients in some it has extended into diastole but not all the way through and in some it has been confined to systole (Fig. 420). Pulmonary hypertension and pulmonary regurgitation is probably more likely to occur in the earlier cases than in PDA. In a review of 21 cases

reported in the literature, one group of writers found a continuous murmur in only 2 (9 per cent). Atypical murmurs—some typical of aortic septal defect. In one case in 11-year-old child operated on by Bailey (39) it was impossible to close the proximal portion of the defect completely because of risk of embolizing coronary circulation. In spite of incomplete closure pulmonary hypertension was reduced and the murmur only systolic before operation because of diastolic and later continuous after operation.

In *pseudotruncus arteriosus*—pulmonary atresia with blood supply to the lungs by the bronchial arteries—a continuous murmur with the pattern of that of PDA is produced in the dilated bronchial arteries which are carrying a relatively large volume of blood (316). The explanation for the continuous nature of the murmur probably is related to the low pressure in the pulmonary circuit—a special case of the phenomenon described by Myers, Murchugh, McIntosh and Blair-dell (1136) (see p. 232). Intratruncal arteriovenous communications when there is large pulmonary flow (288) the finding suggests PDA. This syndrome was found in four of 400 children coming to





FIGS 416 and 417 Congenital thoracic arteriovenous fistula

(above) Gibson murmur heard and recorded in third right inter space of 50 year old female (V J 14562) followed in this hospital for 19 years. The murmur and cardiomegaly have been present throughout but both seem to have been increasing slowly through the years. She has been virtually asymptomatic and pulse pressure is normal. The diagnosis has been aortic stenosis and regurgitation. With the passage of years right axis deviation has appeared and in recent months atrial fibrillation. The developments are not surprising in light of the pronounced dilatation of the right atrium and ventricle on X rays (below) including angiodysplasia (lower right). The vessels involved in the fistula are unknown. A coronary artery to coronary sinus communication is considered likely. A patient (C V 258876 age 67) with strikingly similar story and identical sound recording has been observed at this hospital since 1915.

tens is of the right pulmonary artery (960) is probably closely akin. In the condition the main pulmonary artery is affected. In one series of five cases PDA was also present in four. Grade III or IV systolic murmur at the base of the heart was described. When the systolic murmur especially when accompanied by a thrill is maximal at the right of the sternum aortic tend is also suggested (11081).

In the aortic arch syndromes (1352)—diminished or absent pulses in arteries arising from the arch of the aorta—a continuous murmur (746) may be heard at the base of the neck either on the right (20 903) or on the left (6 213 1136 1794 1423). Particularly in the latter case is IDA simulated. The murmur is not well heard below the clavicle however. Murmurs are heard over the back (203 1304) generated in collateral in this condition which has been appropriately termed reversed coarctation. However the principle outlined by Myer and colleagues (1196) is probably responsible in most cases for the murmur at the base of the neck.

I have been told (961) of a case of *dissected aneurysm of the aorta* with a continuous murmur supposedly identical to that of PDA aortic regurgitation was not present. It may be that this is a special case of the aortic arch syndrome with continuous murmur or the murmur may have been generated in some less well understood manner in the complicated distortion of the aorta.

Venous hums may be very loud in children and readily audible over the upper part of the thorax. Obiteration by pressure on the neck veins and accentuation or attenuation by appropriate manœuvres (see p. 226) usually suffices in identifying the nature of the sound. Furthermore venous hum is usually most intense in diastole and at the right of the sternum is audible at all over the upper chest.

A continuous murmur precisely like that of IDA except for location under the right clavicle has been described in case of total anomalous pulmonary venous return of the figure-of-eight or horse-shoe type (see p. 323).

Usually the pattern of murmurs in AS and AP is quite distinct from that in PDA (fig. 413). I have seen cases of aortic stenosis and regurgitation in which there is a continuous murmur much like

TABLE I

Conditions with auscultatory simulation of patent ductus arteriosus

- 1 Other intrathoracic A-V fistulae
  - a Aortic septal defect
  - b Rupture of sinus of Valvula (a) bulbitis (171) or congenital (91) (161) aneurysm (181)
  - c Artificial (urgic) ductus
  - d Coronary arteries (135 886 1225) to coronary vein (1123) or heart of umbilical (1104) or pulmonary arteries (10) (171)
  - e A-V fistula in the aortic cava congenital e.g. internal mammary (125 1525) traumatic e.g. after rib fracture
  - f Intropulmonary A-V fistula pulmonary artery to pulmonary vein aortic branch (123) lateral pulmonary artery (124)
  - g Subclavian ("A")
  - h Rupture of aortic aneurysm into the pulmonary artery (125 131 17) or superior vena cava (13 170 111)
- 2 Ventricular septal defect
  - a With aortic regurgitation
  - b With pulmonary regurgitation (137)
- 3 Systemic collateral in aortic stenosis (1104 1114)
- 4 Bronchial collateral to lung in pulmonary artery or even pulmonary stenosis (1) (1104 1114)
- 5 Base of aortic valve in aortic stenosis with large pulmonary flow (284)
- 6 Aortic arch aneurysm (131) Multiple congenital tendons of pulmonary arteries (9 47 614 1109 1641)
- 8 Venous hum
- 9 Mammary suff
- 10 Total anomalous pulmonary venous return (1104)
- 11 Aortic stenosis and regurgitation

that of IDA over the lower sternum and axilla and especially the murmur in that area has the same pattern as that of PDA although in the pulmonary and aortic area the appearance and sound is quite typical of double to and fro murmur. In my experience about one in each one hundred cases of aortic stenosis and regurgitation display this phenomenon. The mechanism is not clear. Of course the coarctation of the aorta arch is Charn's network of the right atrium cannot be excluded. Such a lesion can generate a continuous murmur of this type (p. 410).

In late pregnancy and the early puerperium late a continuous murmur resembling the continuous murmur is audible over the upper margins of the

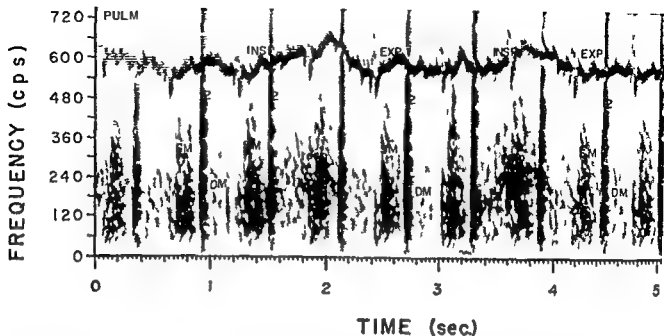


FIG 420 Aortic septal defect

(K 76146) 7 year old boy had always had only a systolic murmur at the left sternal border. There was an apical mid diastolic murmur. ASD was the diagnosis before cardiac catheterization which revealed an oxygen step up in the pulmonary artery. The pressure in the pulmonary artery was 90/54 mm Hg and that in the femoral artery about the same. Yet it was calculated that pulmonary blood flow was about  $2\frac{1}{2}$  times systemic flow. At operation (performed by Dr Henry T. Babinson) the anticipated ductus was not discovered but rather a large aortic septal defect measuring about 18 mm in diameter and larger probably than either the aorta or pulmonary artery. It was possible to close the defect completely. Pulmonary artery pressure was normal after the procedure and the patient has done well.

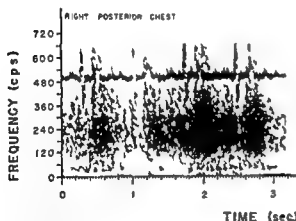


FIG 421 Continuous murmur of bronchial collaterals in tetralogy of Fallot

A 28 year old white male (772277) was clearly demonstrated—by angiography and cardiac catheterization—to have tetralogy of Fallot. Over almost all the right lung field there was a loud continuous murmur identical in quality and timing to that of PDA. Because of clubbing, polycythemia and cyanosis with relatively little incrustation multiple pulmonary arteriovenous fistulas were at first diagnosed but the final conclusion was that bronchial collaterals are responsible for the PDA like murmur.

surgery with the diagnosis of PDA (288). One had a murmur in systole only, the other three had a rather rumbling murmur located in the second and third left interspaces and extending well into but not through diastole.

Rare cases of multiple congenital stenosis of medium sized pulmonary arteries have been reported to have a Gibson type of murmur (29, 422, 616, 1109, 1364). Again, that the murmur in this condition is continuous not merely systolic is explained by the work of Myers *et al* (1136). Eldridge, Selzer, and Hultgren (422) described three cases in two of whom a continuous murmur was heard. These two patients were demonstrated by cardiac catheterization to have a difference in pressure proximal and distal to the stricture during all parts of the cardiac cycle. The case with only a systolic murmur demonstrated little pressure difference during ventricular diastole. Powell and Hiller (1231) reported a long almost continuous murmur at the pulmonary area in a case of pulmonary 'correction'. The syndrome of

TABLE 17

Conditions with an oscillatory auscultation of pulse at the aortic

aortic valve

- 1 Other intracardiac or aortic AV fistula
  - a Aortic septal defect
  - b Rupture of sinus of Valvula (aortic) (b) or congenital (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
  - c Aortic (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
  - d Coronary artery (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
  - e AV fistula in the aortic valve
- 2 Ventricular septal defect
  - a With aortic regurgitation
  - b With pulmonary regurgitation (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
- 3 Systemic collateral in aortic regurgitation of the aorta
- 4 Branchial collateral to lung in pulmonary artery or severe pulmonary stenosis (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
- 5 Subacute (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
- 6 Aortic arch aneurysm (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
- 7 Multiple congenital (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
- 8 Venous hum
- 9 Mammary souffle
- 10 Total anastomosis of pulmonary venous return (a) (b) (c) (d) (e) (f) (g) (h) (i) (j) (k) (l) (m) (n) (o) (p) (q) (r) (s) (t) (u) (v) (w) (x) (y) (z)
- 11 Aortic stenosis and regurgitation

tension of the right pulmonary artery (960) is probably closely akin. In this condition the main pulmonary artery is affected. In one series of five cases PDA was 70 per cent in four. Grade III or IV systolic murmur at the base of the heart is described. When the systolic murmur especially when accompanied by a thrill is maximal at the right of the sternum aortic tension is multiplied (11081).

In the aortic arch syndromes (1112)—diastolic and/or absent pulses in arteries arising from the arch of the aorta—a continuous murmur (746) may be heard at the base of the neck (either on the right (300 903) or on the left (6 213 1176 1301, 1423). Particularly in the latter case is PDA implicated. The murmur is not well heard below the clavicle however. Murmurs are heard over the back (303 1374) generated in collaterals in this condition which has been appropriately termed reversed coarctation. However the principle outlined by Meyer and colleagues (1136) is probably responsible in most cases for the murmur at the base of the neck.

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A continuous murmur precisely like that of PDA except for location under the right clavicle has been described in cases of *total anomalous pulmonary venous return* of the 'figure of eight' or 'non-minor' type (see p. 323).

Usually the pattern of murmurs in AS and AR is quite distinct from that in PDA (Fig. 41a). I have seen one of *aortic stenosis and regurgitation* in which there is a continuous murmur much like

that of PDA over the lower sternum and apical. Graphically the murmurs in that area have the same pattern as that of PDA although in the pulmonary and aortic areas the appearance and sound is quite typical of double to and fro murmur. In my experience about one in each one hundred cases of aortic tension and regurgitation displays this phenomenon. The mechanism is not clear. Of course the acceleration of flow in such a Charnick network of the right atrium cannot be excluded. Such a flow can generate a continuous murmur of this type (p. 410).

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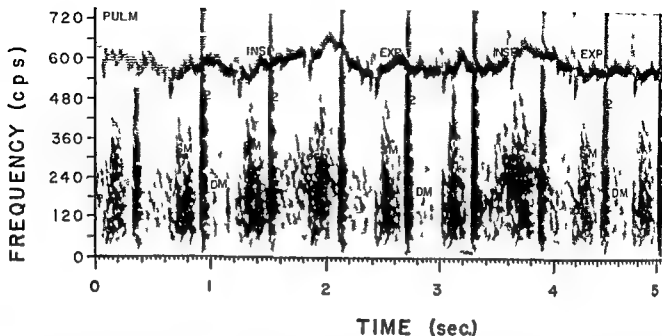


FIG. 420 Aortic septal defect.

C. K. (70456) 7 year old boy had always had only a systolic murmur at the left sternal border. There was an apical mid diastolic murmur (SD) was the diagnosis before cardiac catheterization which revealed an oxygen step up in the pulmonary artery. The pressure in the pulmonary artery was 90/54 mm Hg and that in the femoral artery about the same. Yet it was calculated that pulmonary blood flow was about  $2\frac{1}{2}$  times systemic flow. At operation (performed by Dr. Henry T. Bohnson) the anticipated ductus was not discovered but rather a large aortic septal defect measuring about 18 mm in diameter and larger probably than either the aorta or pulmonary artery. It was possible to close the defect completely. Pulmonary artery pressure was normal after the procedure and the patient has done well.

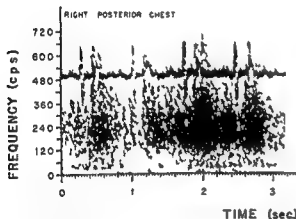


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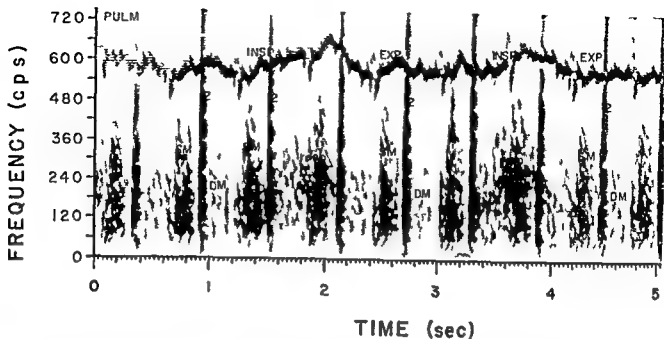


FIG 420 Aortic septal defect

Case (764-6) 7 year old boy had illness had only a systolic murmur at the left sternal border. There was an apical mid diastolic murmur ASD was the diagnosis before cardiac catheterization which revealed an oxygen step up in the pulmonary artery. The pressure in the pulmonary artery was 90/54 mm Hg and that in the femoral artery about the same. Yet it was calculated that pulmonary blood flow was about 2½ times systemic flow. At operation (performed by Dr Henry T Babin) the anticipated ductus was not discovered but rather a large aortic septal defect measuring about 18 mm in diameter and larger probably than either the aorta or pulmonary artery. It was possible to close the defect completely. Pulmonary artery pressure was normal after the procedure and the patient has done well.

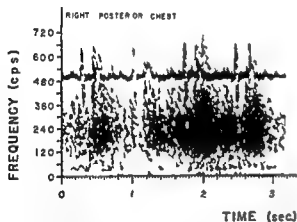


FIG 421 Continuous murmur of bronchial collaterals in tetralogy of Fallot

A 25 year old white male (772277) was clearly demonstrated—by angiocardiology and cardiac catheterization—to have tetralogy of Fallot. Over almost all the right lung field there was a loud continuous murmur identical in quality and timing to that of PDA. Because of clubbing, polycythemia and cyanosis with relatively little incapacitation multiple pulmonary arteriovenous fistulas were at first diagnosed but the final conclusion was that bronchial collaterals are responsible for the PDA like murmur.

surgery with the diagnosis of PDA (288). One had a murmur in systole only, the other three had a rather rumbling murmur located in the second and third left interspaces and extending well into but not through diastole.

Rare cases of multiple congenital stenosis of medium sized pulmonary arteries have been reported to have a Gibson type of murmur (29, 422, 616, 1109, 1564). Again that the murmur in this condition is continuous not merely systolic is explained by the work of Myers *et al* (1136). Eldridge, Selzer and Hultgren (422) described three cases, in two of whom a continuous murmur was heard. These two patients were demonstrated by intracardiac catheterization to have a difference in pressures proximal and distal to the stricture during all parts of the cardiac cycle. The case with only a systolic murmur demonstrated little pressure difference during ventricular diastole. Powell and Miller (1231) reported a long almost continuous murmur at the pulmonary area in a case of pulmonary arteriovenous fistula. The syndrome of

TABLE 17

Conditions with aortic regurgitation as a result of patent ductus arteriosus

- 1 Other intrathoracic abnormalities
  - a Aortic aortic defect
  - b Rupture of sinus of Valvula aortae (syphilitic bicuspid or congenital (91) (165) aneurysm (53))
  - c Artificial (surgical) ductus
  - d Coronary arteries (34, 55, p. 25) in coronary artery (115) or heart chamber (116) or pulmonary artery (116) or (117)
  - e Abnormalities in thoracic cage
    - congenital e.g. internal mammary (118, 119)
    - traumatic e.g. after rib fracture
  - f Intercommunications between pulmonary artery and pulmonary vein aortic branch to peripheral pulmonary artery (48)
  - g Subavian (50)
  - h Rupture of syphilitic aneurysm of the aorta into the pulmonary artery (48, 120, 121) or upper vena cava (122, 123, 124)
- 2 Ventricular septal defect
  - a With aortic regurgitation
  - b With pulmonary regurgitation (13)
- 3 Aortic regurgitation in connection of the aorta
- 4 Bronchial collaterals to lung in pulmonary stenosis or even pulmonary stenosis (125) (126) (127)
- 5 Syndrome of transverse aortic aneurysm with large pulmonary flow (128)
- 6 Aortic arch syndrome (129)
- 7 Multiple congenital stenosis of pulmonary arteries (130, 131, 132, 133, 134)
- 8 Venous hum
- 9 Marfan's disease
- 10 Total anomalous pulmonary venous return (135)
- 11 Aortic regurgitation and regurgitation

stenosis of the right pulmonary artery (960) is probably also taken in the condition the main pulmonary artery is affected. In one series of six cases PDA was present in four. A grade III or IV systolic murmur at the base of the heart was described. When the systolic murmur especially when accompanied by a thrill is maximal at the right of the sternum aortic regurgitation is suggested (1108).

In the aortic arch syndrome (1312)—diminished or absent pulse in arteries arising from the arch of the aorta—a continuous murmur (746) may be heard at the base of the neck either on the right (1008, 900) or on the left (6, 43, 117, 1394, 1422). Particularly in the latter case a PDA is suggested. The murmur is not well heard below the clavicle. However, murmurs are heard over the base (10, 1394) generated in collateral in this condition which has been appropriately termed reversed coarctation. However, the principle outlined by Myer and colleagues (1136) is probably applicable in most cases for the murmur at the base of the neck.

I have been told (901) of a case of dissecting aneurysm of the aorta with a continuous murmur apparently identical to that of PDA aortic regurgitation was not present. It may be that this was a special case of the aortic arch syndrome with continuous murmur or the murmur may have been generated in some less well understood manner in the complicated distortion of the aorta.

Venous hum may be very loud in children and remain audible over the upper part of the thorax. Obliteration by pressure on the neck vein and accentuation or attenuation by appropriate manoeuvre (see 226) usually suffices in identifying the nature of the sound. Furthermore venous hum is usually most intense in diastole and at the right of the sternum if audible at all over the upper chest.

A continuous murmur precisely like that of PDA except for location under the right clavicle has been described in case of total anomalous pulmonary venous return of the "figure-of-eight" or now man type (see p. 143).

It differs in the pattern of murmurs in AS and AR quite distinct from that in PDA (Fig. 11a). I have seen cases of aortic stenosis and regurgitation in which there is a continuous murmur much like

that of PDA over the lower sternum and apically. Crapnell has the murmur in this position have the same pattern as that of PDA although in the pulmonary and aortic area the appearance and sound is quite typical of double to and fro murmur. In my experience about one in each one hundred cases of aortic stenosis and regurgitation display this phenomenon. The mechanism is not clear. Of course the intervention of a lesion such as Churgin's network of the right atrium cannot be excluded. Such a lesion can generate a continuous murmur of the type (p. 110).

In late pregnancy and the early puerperal state a continuous murmur resembling the Churgin murmur is audible over the upper margins of the



congested breasts—the *mammary souffle*—Because of its recrudescence in late systole it appears to be arterial, not venous. It has a superficial quality and its superficial origin is further supported by the fact that it can be obliterated by pressure with the bell of the stethoscope. There is a fickleness about the murmur such that it appears and disappears more or less unaccountably. An intermediate grade of pressure with the stethoscope bell can intensify the murmur or cause one not previously present to appear. The murmur may have a musical quality. Knowledge of the *mammary souffle* (p. 233) and of the characteristics mentioned is sufficient to distinguish this phenomenon from PDA.

The syndrome of VSD with LR may simulate PDA; patients have been mistakenly operated on for presumed PDA (eg. F. R., 720671, 26 year old white female). As suggested previously (p. 362), it is possible that the murmur of the VSD AR syndrome and that of ruptured sinus of Valvula are identical but differ from that of either PDA or of AS AR by features demonstrated in Figure 368.

#### LESS COMMON VARIANTS OF CONGENITAL MALFORMATIONS

Ventricular extension into the abdominal wall—a tube like myocardial cul de sac communicating

with one or the other ventricle, has been described a few times. Parsons (1184) wrote: "A systolic thrill and a continuous murmur could be detected over a smoothly rounded, pulsatile swelling immediately deep to the umbilicus." Powell (1231A) found systolic and diastolic murmurs and thrills in a case of congenital diverticulum of the left ventricle in a 17 year old Bantu.

*Chiari's network* (remnants of the valves of the sinus venosus) occur in the right atrium (259, 594, 1599). It is thought that thrombi sometimes form in the meshes of the *n* networks and result in pulmonary embolism. The only other aspect of clinical significance is the generation of a murmur which may be mistaken for some more grave lesion. Alvarez and Heermann (18) state that in one of these cases "a most peculiar low pitched thronging hum was heard along the right sternal border from the third rib region downward. The hum was musical especially in diastole and faded off into a distant purr in systole." There were similarities to a venous hum except that it persisted with pressure on the jugular veins. Autopsy revealed that the net traversed the inferior vena caval inflow tract. The patient also had syphilitic aortic regurgitation with a conventional murmur. Wilson (1571) described a case in which he thought the murmur of Chiari net resembled the *bruit de Roger*. The patient was a 40 year old man with

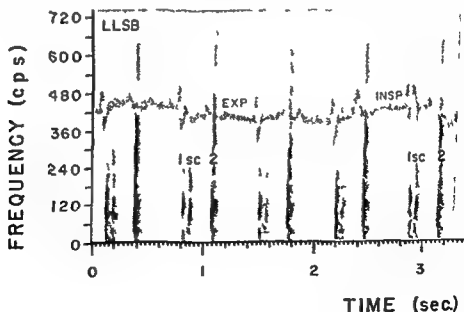


FIG. 422 Truncus arteriosus

I. I. W. (B7470) 5 year old female shows at LLSB an early systolic click, unitary  $S_2$  and absence of aortic murmur consistent with the diagnosis. The sonic findings are probably equally consistent with I/F with pulmonary atresia.

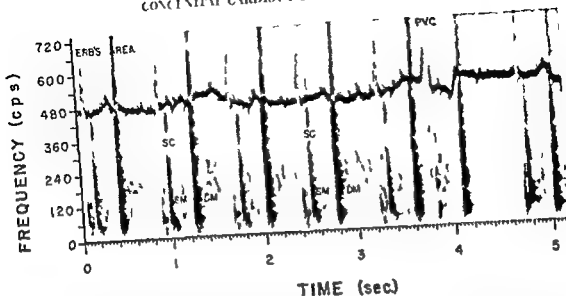


FIG. 423 Truncus arteriosus

When I (1933) A 49 (5) 11 year old male was 4 years old thoracotomy for pre umbilical hernia was performed and he was found to have a true truncus arteriosus. The records show an early systolic click followed by a short systolic murmur which has a pattern consistent with an origin in high flow through the single trunk. There is also a murmur of regurgitation at the arterial valve.

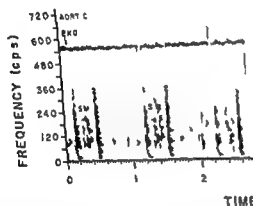


FIG. 424 Anomaly of aortic arch

L B D (4480) 22 year old female has a right sided aortic arch with retroesophageal course. The systolic murmur was transmitted to the interscapular area. The somewhat late onset of the systolic murmur and Christmas tree configuration are consistent with origin in the arch anomaly. In childhood symptoms of origin ring compression incorrectly interpreted as bronchial asthma had occurred.

with malignant hypertension as cause of death. In the last week of life there was a continuous humming, with systolic accentuation at the fourth and fifth left inter space. Of course the location of the murmur was the only point of similarity to the bruit de Roger which is holosystolic, not continuous. It is of note that both patients had a separate

variety of heart disease. Although Christmas tree work is by no means a rare finding at autopsy (being placed at 1.5 per cent by Helwig (1919) and at 2 to 3 per cent by Lister (1928)) the production of an associated murmur probably depends on (1) a proper orientation of the network in relation to venous inflow to the atrium and (2) tension of the network as a consequence of dilatation of the atrium.

#### Truncus Arteriosus Communis (Syn. True truncus)

##### ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

In this malformation a single arterial trunk supplies both the pulmonary and systemic arterial trees. One classification is based on the manner in which the pulmonary arteries leave this common trunk. There is a single arterial orifice and a single arterial valve which according to some should have four cusps for the case to qualify as true truncus arteriosus. There must of course be a large ventricular septal defect or a single ventricle.

Truncus arteriosus is a variety of cyanotic congenital heart disease.

CARDIOVASCULAR SOUNDS. In some cases there are no murmurs. The second sound is unitary as one would expect in view of the single valve. However it may be prolonged and may be followed by

congested breasts—"the mammary souffle." Because of its accentuation in late systole, it appears to be arterial, not venous. It has a superficial quality and its superficial origin is further supported by the fact that it can be obliterated by pressure with the bell of the stethoscope. There is a fickleness about the murmur such that it appears and disappears more or less unaccountably. An intermediate grade of pressure with the stethoscope bell can intensify the murmur or cause one not previously present to appear. The murmur may have a musical quality. Knowledge of the mammary souffle (p. 233) and of the characteristics mentioned is sufficient to distinguish this phenomenon from PDA.

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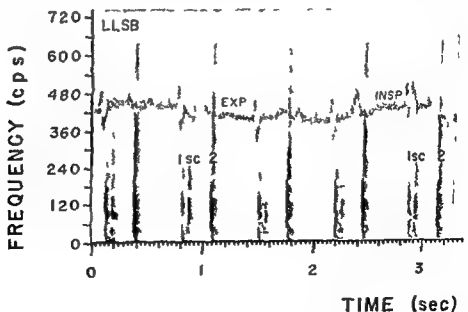


FIG. 422 Truncus arteriosus

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## CHAPTER 17

# Diseases of the Pericardium

**ETIOLOGIC AND ANATOMIC CONSIDERATIONS**  
Beyond the scope of this discussion is a detailed consideration of etiologic factors (1078). The anatomic pattern, use of more pertinent to cardiovascular sound. Acute fibrous pericarditis has many causes but from the standpoint of sound generation there is little significance to anything except the exudation of fibrin on the opposing pericardial surfaces. In the case of purulent pericarditis the fibrin in the exudate is probably responsible for the friction rub. The friction rub in purulent pericarditis has no feature distinguishing it from that of the fibrous pericarditis, such as that of uremia or that of rheumatic fever.

Among the chronic pericardial processes the two of pertinence aside from chronic pericardial effusion are pericardial adhesions and constrictive pericarditis. In the latter condition calcification occurs to a significant extent in roughly half the case. Usually the calcification generalized although it may be denser in some areas than other. Fingers of calcification tend to infiltrate among the myocardial bundles and the calcification may be very dense in the AV grooves.

**PHYSIOLOGIC CONSIDERATIONS** The pathologic physiology of chronic constrictive pericarditis has intrigued clinical physiologists for many years. There is a variety of rather specific physical signs at least one of which is an accentuated and finding by means of special recording (see Fig. 42) which can be considered colligative since all have a common basis in the main physiologic defect—impediment to diastolic filling of the ventricle. It is now evident that constriction of the ventricle is responsible for the clinical manifestation of chronic constrictive pericarditis that is this portion of the heart which must be

deconstricted at surgery and that constriction of the atria and great vessels of limited significance. These colligative phenomena include the following:

1 The protodiastolic sound (Khan's odd diastolic pericardial snap) which is produced by the abrupt halt in filling early in diastole.

2 The flat top and V pattern of ventricular border movement as revealed by electrokymography or roentgenkymography. The bases of this pattern, a simplified one as compared with the normal (see Fig. 42) are as follows:

a The constricted heart is not free to make necessary rotational and configurational changes responsible for the "juggle" during isometric contraction and isometric relaxation.

b Ventricular filling is rapid because of high atrial pressure. The filling limb is usually steeper than the emptying limb.

c Ventricular filling comes to an abrupt halt relatively early in diastole and there is a standstill in ventricular filling during the remainder of diastole.

a The diastolic heart beat (DS") of Wood and colleagues (the Spitznagel's of Khodja) refers to the pattern of the apex beat (or precordial impulse in general) which rather than displaying an outward movement with systole shows a retraction and in early diastole has a sharp outward movement. This paradoxical apex beat has the same basis as the JKL RKL pattern the diastolic heart beat represents a transition to the precordium of the abnormal pattern of ventricular motion. A localized diastolic heart beat is identifiable in a minority of cases in my experience. However in many cases it is possible to note by observing the sternum and left midprecordium obliquely a diffuse systolic retraction and a rapid

a short diastolic murmur. The last feature helps differentiate truncus communis from T/P which it may otherwise simulate (1108A). In some cases there is a loud systolic murmur resulting probably from high flow in the common trunk. In the syndrome of true truncus with large pulmonary flow (288), a continuous murmur identical to that of PDA may occur. There may be an early systolic click produced in the single arterial trunk. See Figure 423.

*Vascular rings* may sufficiently compress or distort the aorta to result in a systolic murmur

heard over the base of the heart and the great vessels. See Figure 424 for such a case.

*Transposition of the great vessels*, including the Taussig-Bing complex (1461), is one of the four most frequent varieties of malformation found in infants. Because of an average prognosis for life of only months, transposition is rare in later childhood and unknown later. The diagnostic findings are not specific. They are the result of oriented malformations (e.g. VSD, pulmonary stenosis) and of changes in the orientation of the great vessels to the heart and front of the chest.

## PLEUROPERICARDIAL FRICTION RUB

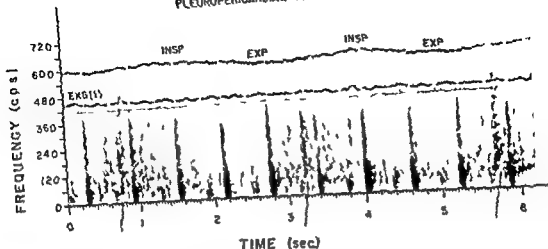


FIG. 4b. Pleuropericardial friction rub

Aortic area in patient with advanced pulmonary tuberculosis. With inspiration a crackle is heard in the first part of systole. There is an inspiratory breath sound running over the heart sound in the region where the crackles are seen. At times pleural and pericardial rub are difficult to distinguish because a the beating of the heart may generate sound in the esophagus and pericardial friction may show pronounced variations with respiration. No evidence of pericarditis in this case.

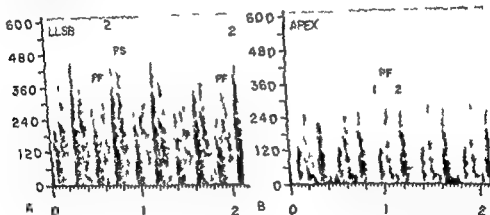


FIG. 4c. Acute tuberculous pericarditis with effusion and moderate cardiac tamponade

C.C. (6131): 7 years of ill tuberculous pericarditis with effusion. At the apex the friction rub is limited to systole. However it is easy to identify it as a rub rather than a murmur because of its quality and displaced later just as the ear can usually make the same identification and because of its circumcribed nature. At the lower left sternal border there is a loud circumcribed sound in protosystole which may be a pericardial friction rub or possibly an early protosystolic sound of the type seen in constrictive pericarditis but also seen in cases of pericardial effusion with tamponade (1074). As is usually the case this protosystolic sound occurs slightly earlier than most protosystolic gallops. However it gives a gallop-like rhythm to the heart sound.

to the confusion which occurs between (1) pericardial effusion and (2) myocardial disease with flabby dilated ventricular wall.

4 The heart sound in pericardial effusion are usually muffled but may remain perplexingly

loud in some cases. Mandant (1030) points out that if the intensity of the heart sounds when the patient is on his knees and elbows is compared with their intensity when he is on his back, muffling of the heart sounds will be demonstrated

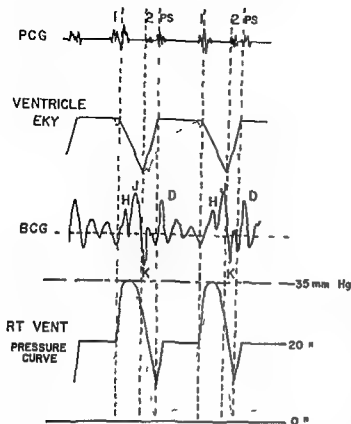


FIG 42a Chart correlating four early diastolic phenomena of constrictive pericarditis (1) early diastolic sound (ps) in the phonocardiogram (PCG) (2) flat top and V pattern of ventricular border movement as displayed by the electrokymogram (EKG) or roentgen kymogram (3) the abnormally large diastolic excursions (D) in the ballistocardiogram (BCG) (4) the early diastolic dip followed by diastolic plateau of the right ventricular pressure curve

outward movement in early diastole. External pericardial adhesion without the constriction syndrome can produce a simulating pattern.

4. The right ventricular pressure curve obtained by cardiac catheterization shows (rather than the usual gradual rise of pressure in diastole through the range of only a few mm Hg) an early diastolic dip followed by a diastolic plateau. Early in diastole the pressure in the ventricle falls to a level approaching zero, but rises abruptly to end in a diastolic plateau at a high level pressure. Obviously, this level is essentially the same as that in the peripheral veins, i.e. as much as 25 mm Hg in many instances. In fact, it is sufficiently high that one may speculate whether the pulmonary valve might reopen during late

diastole because the pressure in the right ventricle exceeds that in the pulmonary artery. This could probably happen only when the left ventricle is relatively free of constriction, otherwise diastolic pressure in the pulmonary artery is likely to be elevated.

5. The "water hammer" phenomenon when the capacity of the ventricle is attained in diastolic filling is responsible not only for the protodiastolic sound, the mid or turning point of the early diastolic dip, and the end of the "V" of the "flat top and V" pattern and of the outward movement of the diastolic heart beat, but it also produces abnormal early diastolic excursions in the ballistocardiogram. In side to side ballistocardiograms these are most striking, seemingly indicating that the force involved operates not only in the lateral direction.

Any laceration of the heart and diffuse myocardial fibrosis as well as endocardial sclerosis (so called "constrictive endocarditis" (107)) may so alter the compliance, and therefore the pressure-volume characteristics of the ventricle that physiologic and clinical simulation of constrictive pericarditis results.

**CARDIOVASCULAR SOUND.** The friction rub of acute pericarditis has been fully described (see p. 223) its transitory nature, the necessity of listening in many areas in several positions of the patient on numerous occasions in cases of suspected pericarditis, the triple character of the friction rub in full blown form but the isolated systolic or occasionally diastolic timing during early and late stages of its evolution, the occasional musical quality like a wet finger rubbing on glass, the possibility of confusion for a to and fro murmur of aortic stenosis and regurgitation.

Pericardial friction rub may occur with pulmonary emboli (112). It is the pleural surface that is rubbed by the beating heart. There is usually, although not invariably, respiratory variation in the pericardial friction rub of this type which in actuality is a pleuropericardial rub (Fig. 426).

With pericardial effusion and tamponade there may occur a protodiastolic sound which probably has the same mechanism as that of constrictive pericarditis (36). This sound endows the heart with a gallop rhythm (Fig. 427) and contributes

\* That in the left ventricle is shown the same pattern whenever recorded.

## DISEASES OF THE PERICARDIUM

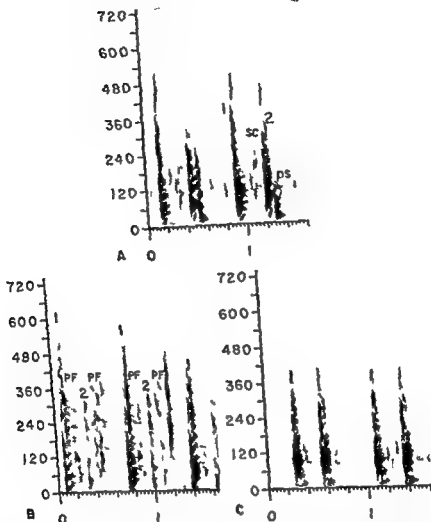


FIG. 490. Sound in calcific constrictive pericarditis.

**A** I P (65 yr) aged 100 years. Note the aortic click (sc) produced by the calcified plaques, the light splitting (ps) in the second cleth, the early diastolic sound (1-2) which simulates the second cleth to create a false impression of widely split S<sub>2</sub>. **B** Pericardial friction rub, early after pericardiectomy for constrictive pericarditis (I I P 65 yr). **C** Later after operation at which time only light rattling sounds in systole and diastole persist.

**CONSTRUCTIVE PERICARDIUM: SIGNS OF PERICARDIAL DISEASE** Although well-documented in the world literature there is not general familiarity with several of the puzzling auscultatory phenomena of pericardial origin.

Pericardial adhesions can produce a systolic click and pericardial roughening can produce a systolic murmur. Not infrequently the two phenomena are associated; the systolic click introducing the murmur. The murmur of pericardial roughening is never holosystolic. The murmur may be noisy or less frequently musical. The systolic click of pericardial adhesion is

usually mid- or late systolic. When in late systole it produces with the second sound a combination suggesting second sound plus opening snap of mitral stenosis—especially since such a late systolic click may develop after acute rheumatic fever with pericarditis. The systolic murmur frequently occurs in the same clinical setting, i.e. after rheumatic fever and produces its own confusion—for the murmur of mitral regurgitation. The musical late systolic murmur of pericardial roughening can be very loud and disturbing as a possible indicator of grave heart disease—which it is not (see p. 207).



even with an effusion of small size. Normally, the intensity of the sounds is unchanged or increased in the knee elbow position.

Colvin (280A) found, in cases of pericardial effusion, electrical alternans and auscultatory alternans without pulsus alternans. He suggested that effusion increases the mobility of the heart permitting pendular motion of the heart with a natural period of oscillation related in a 2:1 manner to the heart beat.

Usually a pericardial friction rub tends to disappear as pericardial effusion develops. However, the presence of a friction rub does not exclude the possibility of there being present a considerable amount of pericardial fluid.

When air as well as fluid is present in the pericardial sac, the so called *bruit de moulin* or mill wheel sound results. This situation is rarely seen these days except on an iatrogenic basis the air either being inadvertently introduced at pericardial paracentesis or being introduced intentionally for purposes of radiologic study of the heart and pericardium. The mill wheel sound was so named (*bruit de moulin*) by Bricheteau (174) in 1844. The Geimans use the corresponding *Muhlen geräusch* (712). In 1924 Stahl and Entzweig (1431) described it when they removed fluid from the pericardial sac in a patient with tuberculous pericarditis and replaced it with air.

Bizarre sounds—often very loud—have been reported following perforation into the pericardial cavity by carcinoma of the esophagus (1144), by peptic ulcer of the esophagus (339) or stomach (619), by trauma occurring as an occupational hazard of sword swallowing (1182), or from foreign bodies, (such as mutton bone in the case reported by James (743)). Rupture of the esophagus into the pericardial sac, either through perforation of a peptic ulcer or penetration by a foreign body, produces a characteristic syndrome with severe substernal pain and shock and with a peculiar precordial murmur as an important feature. In 1849, Parke (1182) recorded the melancholy case of a 19 year old sword swallower who some hours before death from this condition developed a friction sound both diastolic and systolic mixed with a peculiar kind of metallic rhonchus carried up to the top of the sternum. Gellman and Silberstein (339) described the case of a 63 year old man with previous history of duodenal ulcer and sliding diaphragmatic hernia. He died 21 hours after onset of pain. There was a peculiar precordial murmur which could not be separated from the heart beat. One had the impression that liquid was rushing through a narrow opening. Recovery with surgical intervention is possible in these cases if the diagnosis is made promptly.

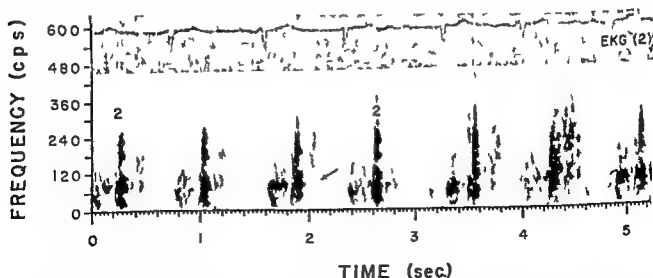


FIG. 428. Extracardiac early diastolic murmur or pulmonary regurgitation?

W. W. (494637) 82 year old male has no specific illness other than generalized arteriosclerosis and emphysema. There is a variable early diastolic murmur in the pulmonary area. Stethoscopically and phonocardiographically there is a suggestion of murmur. The characteristics of the murmur are compatible with pulmonary regurgitation as described by Holldack and colleagues (699-1120). Bourne described stethoscopically what seems to have been the same murmur and interpreted its origin as extracardiac.

**CHRONIC CONSTRICTIVE PERICARDITIS** The most characteristic single auscultatory finding is an early protodiastolic sound (930). It occurs closer to the second sound than a protodiastolic gallop (Fig 429) and is sometimes confused for a split second sound. When the phenomenon called diastolic heart beat is present it is noted that the protodiastolic sound is temporally related to the end of the sharp outward movement of the precordium. The higher the venous pressure, the more rapid is ventricular filling and the closer is the adventitious sound to the second sound. In cases of early constrictive pericarditis there may in fact be variation in the S<sub>2</sub>-extra sound interval, this measurement being least at the end of inspiration. The protodiastolic sound is likely to be louder and more clicking when there is extensive calcification in the pericardial scar. It frequently exceeds the first and second heart sounds in intensity.

The first heart sound is usually but not invariably diminished in constrictive pericarditis. It is frequently accentuated, however, because a certain amount of pulmonary hypertension due to left-sided constrictive pericarditis is frequent. That all the heart sounds are not more regularly suppressed may be related to the improved coupling between the heart and thorax in these cases.

Multiple clicks in systole are usually heard in cases with extensive calcification. There may be a single click in the first part of systole about 0.11 sec after S<sub>1</sub> (400).

After pericardectomy several changes of note may occur in the auscultatory findings: (1) A mid-diastolic murmur may develop. (2) The protodiastolic sound is attenuated or disappears. If still present it occurs later after S<sub>2</sub>. Dunn and Dickerson (382) using the Q-S<sub>2</sub> interval found a value of 0.47 before surgery and 0.22 after surgery (see Fig 84). (3) A split second sound frequently occurs and S<sub>1</sub> may also be split. (4) S<sub>3</sub> diastolic and diastolic rubbing sound suggesting S<sub>3</sub> diastolic and diastolic murmur may be heard.

In three patients (two (E S and W C) teen age males and the third (F G) a female in her twenties) I have observed after pericardectomy a third heart sound followed by a short rumble. Functionally the patients are well. However they have moderate cardiac enlargement. Ven-

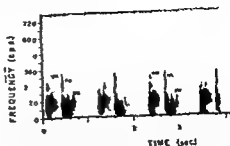


FIG 430 Carey Coombs murmur after pericardectomy for tuberculous constrictive pericarditis in 1 S (3231). The heart remained dilated after operation. The murmur had not been heard before operation.

ticular dilatation without common valve enlargement of the AV orifices; possibly the basis of the murmur. I have not heard the murmur in question before operation. The murmur can lead to a mistaken impression of a so-called mitral stenosis. Probably rheumatic fever rarely, perhaps never, causes constrictive pericarditis (719) (76); the incidence of a rheumatic valve lesion and constrictive pericarditis in combination can be expected to be the product of the incidence of the individual condition—a small value. The possibility exists that some of the cases reported clinically as instances of a so-called mitral stenosis and constrictive pericarditis in patients with this particular type of diastolic murmur on the basis of constrictive pericarditis alone. Levine (88a) points out that occasionally a faint prolonged third heart sound is audible at the apex resembling a faint murmur of mitral stenosis. Jackson (740) described a patient in whom the murmur of mitral stenosis became evident after pericardectomy. Whether the patient had true mitral stenosis is unknown. Burwell (201) tells me of an autopsied case of the ascription of tuberculous constrictive pericarditis and rheumatic mitral stenosis. White and co-workers (1511) described a series of cases with predominantly left-sided constrictive pericarditis. The first of the patients was said to show pressure on the mitral orifice by

\* I (1099, 10 S) have never encountered constrictive pericarditis in a case of substantial rheumatic valvular heart disease. Such cases I never have reported (318, 46); the cases may represent concealed however clinically atypical mitral stenosis pericarditis without constrictive pericarditis. Some time found in a connection with valvular heart disease.

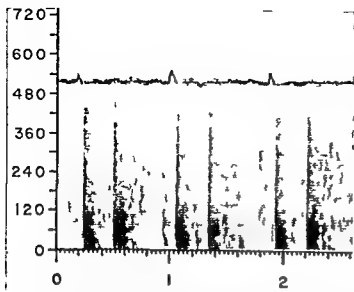


FIG 430 Large precordial thoracic cage defect

In this patient (J H 40330a) the costal cartilages and a large portion of the body of the sternum were removed in the several stages of cardiac decortication for constrictive pericarditis. The last operation was performed in 1931 and the patient is now free of symptoms of cardiac compression. The heart lies immediately beneath the skin. In most areas of the precordium sounds suggesting pericardial friction sounds or even endocardial murmurs are audible. It is these which are displayed here. The rub has three phases: systolic, protodiastolic and presystolic. Residual roughening of the surface of the heart and its superficiality are considered to be the factors responsible for these sounds.

There is essentially a syndrome (222-932 p 210) consisting of sticking pericardial pain, palpitations and cardiac neurosis in association with pericardial clicks and murmur. Friction of the heart on pericardial adhesions may be responsible both for the sticking pains and for the palpitation in terms of heart consciousness. The neurosis is often idiopathic to a considerable extent. Because of ignorance of the true nature of the auscultatory findings the physician attaches more significance to the finding than it deserves. Furthermore, in the electrocardiogram residual T wave changes from previous pericarditis may be misconstrued as indicating coronary artery disease. See Figure 431. Discovering characteristic systolic clicks assists identification of chest pain as pericardial, not coronary arterial, in origin.

Diastolic musical murmurs of pericardial origin also occur. I have encountered them after craniotomy for mitral valvulotomy and without such intervention in patients with large hearts on the

basis of rheumatic disease. In these patients not only is there probably pericardial roughening but also the large heart rubs on its surrounding structures more than the heart of normal size.

Occasionally I have encountered noisy murmurs in diastole which I thought because of their quality and timing and because of the absence of valvular heart disease were probably of extracardiac origin, specifically pericardial roughening. For years following decortication for chronic constrictive pericarditis noisy diastolic murmurs on this basis may be audible (see later) and in individuals without cardiac surgery a similar murmur is occasionally heard. Figure 428 presents the recording from the pulmonary area of an 81-year-old man who had a diastolic murmur seemingly on this basis. (Origin at the pulmonary valve cannot be excluded. With normal pulmonary artery pressure the murmur of pulmonary regurgitation is thought to demonstrate this pattern at times. Fenestration of the valve (p 273) is possible.)

Melik Gulnarayan (1992) pointed out that the broad category of pericardial friction rubs includes both extra- and intrapericardial varieties. It is usually difficult to distinguish the two. When extrapericardial in origin, the rub is likely to display more respiratory variation.

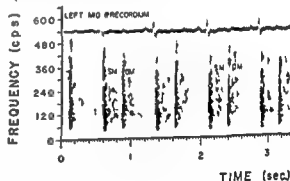


FIG 431 Simulation of endocardial murmurs for years after pericardectomy

Recorded at the area indicated, not over the defect in M C (365311) in whom pericardectomy for constrictive pericarditis was performed nine years previous. The patient has been functionally cured since operation. Frequently after operation an impression of mitral stenosis was recorded because of an early or mid diastolic rumble at the apex. At times even a presystolic murmur was described. The patient has a large defect in the left anterior thoracic rib cage; forceful pulsations of the heart are evident in this area.

## CHAPTER 18

# Diseases of the Myocardium

**PHYSIOLOGIC CONSIDERATIONS** The etiologic factors in this group of disorders are too numerous and varied to discuss in detail. Changes deriving from coronary artery disease, the most frequent basis for infarction of the heart and diffuse myocardial fibrosis, are pertinent to cardiac sound because the physical properties of the heart muscle are so altered that its behavior is strikingly similar to that of the heart in constrictive pericarditis.

**CARDIOVASCULAR SOUND** At least three characteristics of the sound in myocardial disease are noteworthy: (1) dull heart sound, (2) gallop, (3) pulsus alternans with alternation in the intensity of the heart sound and of murmur.

Dull heart sound are characteristic of myocardial disease but may be difficult to distinguish from the damped sound of pericardial effusion. The dull second sound is explicable on the basis of low blood pressure. When the first heart sound is of poor quality, it is probably related to the relatively slow contraction of the ventricle with lower than normal development of intraventricular pressure. Therefore the action resembling a sound the snapper is the sound. The converse is also true. Abnormally low myocardial contraction in the pre-systolic contraction period is suggested by prolongation of the Q-T interval in cases of myocarditis.

There is unfortunately a large element of arbitrariness involved in judging of the quality of the heart sounds. A heavy muscular obese or emphysematous thorax may also result in heart sound of poor quality.

The first sound is often impressively attenuated in adolescents or children with myocardial involvement as part of progressive muscular dystrophy or Friedreich ataxia. The finding is of particular note in the younger age groups.

Kuhn and Holdick (522) described an attenuated first sound in 12 of 16 patients with myotonic dystrophy.

It is the protodiastolic gallop which is characteristic of myocardial disease although with advancing heart failure a presystolic gallop may also be present. Gallops are not easily heard by the novice. Detection of faint gallops probably requires more experience than any other of the myocardial culture phenomena. Yet the clinical importance of the phenomenon is an indication of the state of the myocardium makes it very important that the clinician learn to recognize it. The auscultatory characteristics of gallop have been detailed earlier (see p. 174).

See Figure 413 for description of the case of a 27-year-old white male in whom extensive myocardial fibrosis from coronary artery disease was accompanied by atrial fibrillation, snapping  $M_2$  and a mid-diastolic rumble apparently on the basis of dilated left ventricle. These auscultatory features led to the diagnosis of mitral stenosis.

**Pulsus alternans** occurs occasionally in myocardial disease. It is best detected with the blood pressure cuff and always should be quantitated in terms of mm Hg difference in systolic pressure between alternate beats. Pulsus alternans may be present only in the recumbent position. Probably the reduction in venous return in the upright position helps the competence of the heart—in effect venoconstriction or tourniqueting is performed. On the other hand, Friedman, Dault, and Sheffield (484) found pulsus alternans in certain hypertensive only in the upright position. Or if the patient was recumbent venous pooling with tourniquet or the Val-salva maneuver brought on pulsus alternans.

With pronounced pulsus alternans the heart sounds in alternate cardiac cycles may show it

the calcified pericardium "resulting in definite mitral stenosis." In the 1890's Fisher (461) and Phear (1205) described a series of patients with a mitral diastolic murmur and neither organic mitral stenosis nor aortic regurgitation, many of the cases had what was then referred to as adhesive pericarditis.

The confusion of constrictive pericarditis with rheumatic heart disease is always possible because in the former condition atrial fibrillation occurs in about one third of cases, cardiac enlargement, often marked, is present in over half (contrary to the "small, quiet heart" emphasized by earlier writers) the protodiastolic click may suggest a mitral opening snap, P is frequently accentuated, the left atrium may be quite large from irregular distribution of constriction, and, of course, the subjects may be young.

The dilation of the ventricle present postoperatively in the patients with a diastolic murmur after operation is probably on the basis of disuse atrophy—the heart has, in essence, been in a plaster cast for an appreciable period. Acute dilation of the heart after operation may occur is sometimes fatal and probably is often damag-

ing. Digitalization immediately after operation or even before operation is, in my opinion, indicated in such cases.

After operation the early protodiastolic clicking sound may disappear or may be replaced by a third heart sound conventional in timing and quality. The third heart sound may persist only during the period of cardiac dilatation in the first days or weeks after operation or may remain more or less permanently, as in the case illustrated in Figure 432, if the ventricular dilatation persists. Many cases do not show a third sound at any time.

In some cases, especially those operated on early in the evolution of the surgical technique for constrictive pericarditis and especially those cases which required more than one operation because of inadequate decortication the first time, there are systolic and diastolic friction sounds which can easily be confused for systolic and diastolic murmurs (see Figs. 430 and 431). These are usually patients who have a large precordial defect in the bony thorax. However, the "murmurs" in question are audible over intact portions of the chest as well as over the defect itself.

ternation in intensity and there may be alternation in the intensity of murmur. In one study (484) 8 of 11 patient with pulsus alternans showed heart sound alternation as well. In even cases of pulsus alternans another group (49) found concordant alternation of the first sound in all cases concordant referring to louder sound related to stronger radial pulse. In four there was also alternation of the second sound one concordant three discordant. Ivan and Racine (93) described alternation of aortic and mitral murmurs during left ventricular failure.

It is well recognized that with myocardial disorders regurgitation at the mitral or the tricuspid valve or both may develop.

In one patient (Γ Α 31439) a 50-year old colored male with what eventually was proved at autopsy to be diffuse myocardial fibrosis presumably as the result of myocarditis a long rough grating, leathery systolic murmur at the left of the sternum in connection with the EKG changes displayed in Figure 134 led to the

diagnosis of septal perforation following myocardial infarction. The murmur was accentuated by inspiration and there was a systolic pulsation of the liver. It is possible that the murmur was produced by relative tricuspid regurgitation although in alternative possibility a pericardial origin. A murmur of pericardial origin would be likely to show similar respiratory variation. Autopsy revealed that the right ventricle was affected especially severely with almost paper thin state of it wall.

*Myocardial infarction* (1018-1183) results in heart sound of poor quality diastolic gallops a pericardial friction rub (pericarditis epicarditis) a murmur of mitral regurgitation from dilation of the left ventricle or infarction of a papillary muscle with or without rupture of aortic aortic click or thrust from aortic expansion of a ventricular aneurysm. During the acute stage a pericardial friction rub occurs frequently—in 20 per cent of the 50 cases in one series (1286) one-third of cases in another (122) and a minority (8)

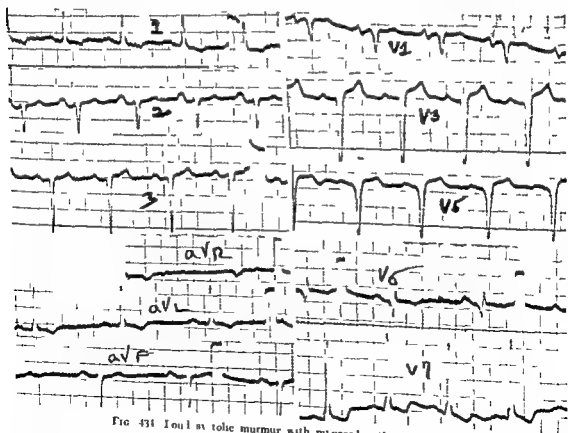


FIG. 431 Loud aortic murmur with myocardopathy (see text)

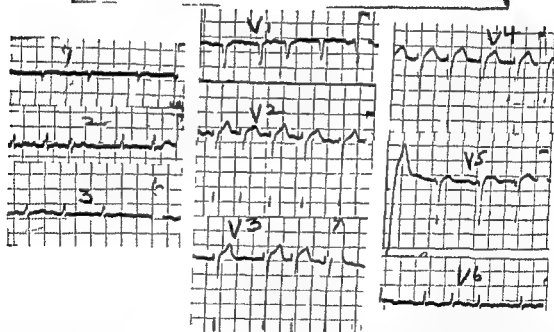


FIG. 433 Diastolic rumble simulating mitral stenosis in patient with myocardial infarction, from coronary artery disease

G. B. (752086) aged 27 years was diagnosed as having rheumatic heart disease with atrial fibrillation, mitral stenosis and mitral regurgitation. He was a strapping healthy looking white male weighing 228 pounds. A faint apical diastolic thrill was described.  $S_2$  at the apex was described as snapping. There were a grade I apical systolic murmur, no opening snap, and a late diastolic murmur which varied in length and intensity with cycle length. Another observer described what he interpreted to be either a third heart sound or an opening snap. The diastolic murmur was increased in the left lateral decubitus position. Electrocardiogram showed bizarre pattern of the QRS complexes with low voltage and no R waves of appreciable size in any lead. On left heart catheterization the left ventricle could not be entered from the left atrium. Pressure in the left atrium is very high (40/28(?) mm Hg) and the shape of the curves suggested mitral regurgitation. At operation no stenosis was found. At autopsy a few days later the mitral valve was perfectly normal, the left ventricle was much dilated, there was extensive coronary atherosclerosis and myocardial fibrosis secondary thereto.

acute coronary occlusion. In one patient a soft high pitched blowing presystolic murmur was heard in the fourth inter-space just to the right of the sternum from the 15th to the 23rd day of illness. In a second patient a murmur of the same timing and quality was heard over the sternum and to either side at the level of the third fourth and fifth inter-spaces. That this was an atrial friction would seem most likely. The author thought however it arose through some interference with the tricuspid valve.

The complications or sequelae of myocardial infarction which are accompanied by auscultatory findings of note are pericarditis, ventricular aneurysm, rupture of the ventricular septum (1024) and rupture of a papillary muscle. The last accident is most likely to involve the left posterior papillary muscle and to occur with occlusion of the right coronary artery.

Perforation of the interventricular septum may occur within less than a day of the onset of symptoms (1338). In 1948 Lower and Inley (477) found 36 cases of ruptured ventricular septum in the literature and added two cases. Among the 38 of these 38 cases in which length of survival was known 43 lived less than one month and 39 less than one year. Occasional patients live longer (733, 791) one remarkable patient survived for 8 months (1607). Of 46 cases examined 44 showed a systolic murmur which was usually maximal to the left of the lower sternum. In 2 it was accompanied by a thrill. Three showed a diastolic murmur as well. Large size of the defect was thought to account for the absence of murmur in one case (100) and profound shock in the second (73). (Non-penetrating trauma to the chest is occasionally the cause of perforation of the septum (1420).)

The following features of rupture of a papillary muscle help differentiate it from rupture of the interventricular septum (1338): (1) the murmur is more bizarre often diastolic and loudest at the apex; (2) a thrill is usually absent; (3) acute left ventricular failure is more likely to develop; (4) the murmur is absent surprisingly often. Aker (75) pointed out that a murmur is described in less than half the reported cases and emphasized the absence of thrill as a rule. He also was impressed in his own experience and that reported

in the literature with the incidence of 'pseudo-rub'. He thought it in fact to be a murmur with an unusually superficial quality.

Sunder et al (1311A) concluded that it is most often the posterior papillary muscle which is ruptured and that the location of the infarct is most often posterior. Septal rupture is more likely to occur with anterior infarction.

In a case of ventricular aneurysm following myocardial infarction we (1957) found an early systolic click. Mandam (1050) has had a similar experience. A mid-systolic click has also been described (498, 852).

With ventricular aneurysm a strong parasternal pulsation sometimes localized to the region of the normal apex beat sometimes more diffuse with paradoxically weak peripheral pulses and distant heart sound is characteristic. Systolic and diastolic murmurs heard in a localized area usually at the site of the pulsation have been described by many observers but are perhaps still too little known. Among those who have emphasized this finding are Schaff and Brook (1336) and Paul (1186). Henslinger (1260) found a *rumal quality* to both the systolic murmur which sounded like *ou* and the diastolic which sounded like *e*. In five of 20 cases of ventricular aneurysm Auld (1183) found a fairly loud and long murmur with a peculiar



FIG. 436 Ventricular aneurysm in which was a characteristic unusual murmur. (See text.)



per cent in still other series (72). The rub occurs usually in the first week and most frequently in the second or third day. It may be heard with posterior myocardial infarction as frequently as with anterior infarction (1443), the pericarditis with any large infarction may be generalized. An unusually prolonged pericardial friction rub should arouse suspicions of hemopericardium, a complication which occurs without anticoagulant therapy (20), but is more likely to occur when this therapy is used. Organization of the exuded blood may be the basis for a systolic murmur, usually limited to late systole, after myocardial infarction. Recurrent pericarditis with chest pain and friction rub, occurs in some patients after myocardial infarction without any repeat infarction.

In 28 per cent of cases of acute myocardial infarction Shillito and others found a diastolic gallop (1386).

Castex (245) of Argentina described six cases of anterolateral myocardial infarction accompanied by a circumscribed systolic murmur in mid or late systole. He suggested that the murmur is produced by the roughening of the endocardial surface—an untenable view in light of present analyses of the physics of cardiovascular sound. It is likely that the murmur Castex described was of pericardial origin. He quoted Huehard (see p. 19) as considering a murmur of this type as a sign of cardiac aneurysm.

Wolferth, Wood and Mugholies (1580) described an auriculo-systolic murmur in the 'tricuspid area' in two patients concluding from

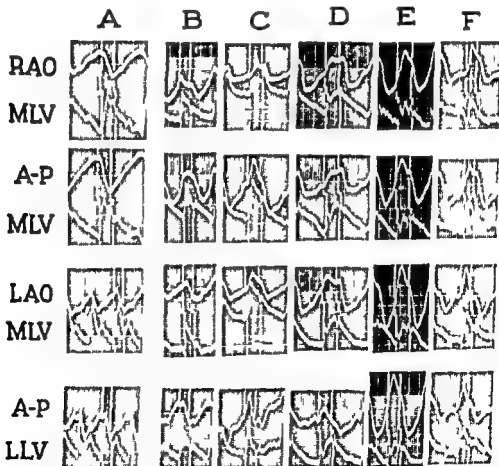


FIG. 43. Electrocardiograms (above in each recording) from the ventricular border in a normal subject (A) and in four patients with myocardial infarction (B to F). The carotid pulse (below in each recording) is used for timing purposes. In the LLV a downward excursion indicates an inward movement of the heart border and an upward excursion an outward movement. The point of importance is that the ventricular wall affected by an infarct tends to show an outward movement in systole whereas in early diastole it moved inward. This is the familiar paradoxical motion of a ventricular aneurysm which may be present in milder form as a dynamic aneurysm as it were. RAO, right anterior oblique; of the patient; AP, antero-posterior; LAO, left anterior oblique; MLV, mid and lower left ventricular border, respectively.

## CHAPTER 19

# Systemic Arterial Hypertension

The second sound at the base is accentuated in systemic arterial hypertension. Usually  $S_2$  is louder than  $P_2$  but occasionally the opposite is true. Since the pulmonary second sound has contributions from the closure sound of both the aortic and the pulmonary valves whereas the aortic sound is of unitary origin it is not difficult to understand the seeming paradox of  $P_2$  louder than  $A_2$ . The latter phenomenon is most frequent in young persons.

The second sound is not split in hypertension. Contrary to what seems to be generally thought hypertension of either circulatory systemic or pulmonary without bundle branch block or heart produces no impressive or consistent splitting of the second sound.

The mitral closure sound is usually delayed in systemic arterial hypertension. The Q1 interval is prolonged although it is not as long as in most mitral stenosis (An aortic regurgitation sound must not be confused for the mitral closure sound). The mechanism of the prolonged Q1 is not entirely clear. Possibilities are the following: (1) Through effects of hypertension on the myocardium contraction may be slower during the pre-isometric contraction phase. In myocarditis the Q1 interval is indeed likely to be prolonged. (2) Because of premature transmission of the atrial pressure wave to the ventricle as suggested by the features of the presystolic gallop outlined on p. 179 the normal reinforcement of A valve closure may be lacking in hypertension. A delay in mitral closure might occur for a reason similar to that for the delay in mitral closure in atrial fibrillation and with a prolonged PR interval.

The first sound may seem to be split because of the occurrence of an early presystolic click and of course the presystolic gallop may with  $S_1$  produce a combination suggesting a split first sound.

Presystolic gallop is characteristic of hypertension (1916).<sup>1</sup> Although the connecting link was not then appreciated it was the association of presystolic gallop with hypertension which led to its designation as *bruit de brightique*—sound of Bright's disease—by the French a century ago. See page 174 for a detailed description of the auscultatory characteristics of the presystolic gallop.

Occasionally the pulmonary hypertension resulting from chronic left ventricular failure in systemic hypertension is of such proportions as to result in a Graham Steell murmur. In one patient (W. A., 1906/41) in whom this was the case the Graham Steell murmur showed accentuation with inspiration.

Relative aortic insufficiency from dilatation of the base of the aorta occurs rather frequently in arterial hypertension. It is difficult to estimate the frequency (Arvin (1926) placed the incidence at 14 in 200 autopsied hypertensives—7 per cent. White (1949) cites the figure 4 per cent. Amyl nitrite may eliminate the aortic diastolic murmur of this causation but it is doubtful that the effect is specific. Aortic regurgitation may occur if there is dilatation of the base of the aorta due to cystic medial necrosis secondary to the hypertension (or due to syphilis or the Marfan syndrome). Obviously it is also likely to occur if there is an associated congenital bicuspid aortic valve or a rheumatic lesion of the valve which of itself might produce little or no murmur.

<sup>1</sup> It is shocking to find the author of the monograph on hypertension equating presystolic gallop to split first sound (1966). The only justification is the auscultatory similarity. The recent demonstration of Leonard and colleagues (1968) that a normal atrial contribution to the first sound may be picked up by presystole and constitute the presystolic gallop of hypertension (see p. 196) provides gratifying justification.

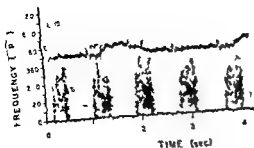
'cooing,' 'plutiv' or musical character, occupying the whole of systole and early part of diastole, best heard at the site of the cardiac impulse, and not conducted in any particular direction." Three possible mechanisms for this musical murmur—as well as the noisy ones—exist (1) mitral regurgitation, (2) flow of blood in and out of the aneurysm, (3) pericardial friction.

If the patient survives, the murmurs may diminish possibly because the aneurysm fills with clot. Scherf and Brooks (1936) have emphasized a pre-systolic accentuation of the diastolic murmur. Mitral stenosis has been mistakenly diagnosed in these cases. In the phonocardiograms presented by Scherf and Brooks (1936) the systolic murmur was in the first part of systole and the diastolic murmur followed the second sound immediately. These findings are consistent with the view that the systolic murmur is produced by the expulsion of blood from the main ventricular cavity into the aneurysm, and the diastolic murmur by the aneurysm bleeding into the ventricle, i.e., emptying itself into the ventricular cavity when the pressure in the ventricle falls below that in the aneurysm (see Fig. 435 for EKG demonstration of this pattern of ventricular

border movement). This mechanism finds difficulty in accounting for the pre-systolic accentuation. Actually, the phonocardiographic evidence for pre-systolic accentuation is not too convincing.

Auscultatory changes with cardiac rupture have been described by Reznikoff (1937), Massey and Drake (1946, 1947), Nuzum (1948) and Bishop and Logue (1966). In a patient in whom rupture occurred during auscultation, Massey and Drake (1946) stated that "no friction rub was heard before this spectacular episode but immediately after its inception an intense grating pericardial rub was audible from the third left inter-space down to the apex."

In a recent patient (G. B., 763681) with anterolateral myocardial infarction it was noted that the apex beat had an unusually large outward excursion early in systole, that in contrast the heart sounds were feeble and that a localized bulge was present at the lower left heart border on x-ray. The patient died from rupture of a ventricular aneurysm (Fig. 436) 12 days after admission. About 24 hours before death the apical pulsation was observed to be much more striking and a systolic murmur and early diastolic murmur of unusual quality were heard.



Considerably more men tricuspid regurgitation is accompanied by release of thrombotic material in some women

Any pulmonary hypertension which may be present is exaggerated by the presence of high flow in the pulmonary circuit—be it transient as with exercise or permanent as with septal defect

The pulmonary valve snap is less infrequent

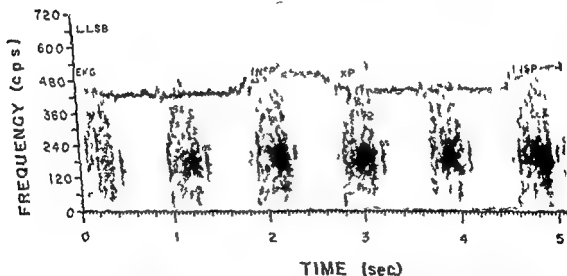
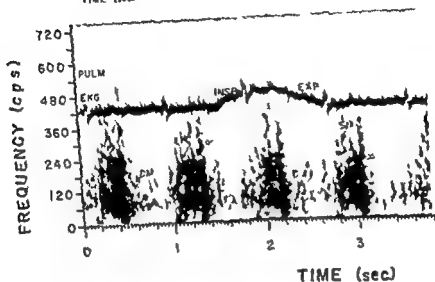


Fig 478 Similar to opening snap in primary pulmonary hypertension

A 36 year old white female was thought clinically to have primary pulmonary hypertension. The clinical condition was confirmed at autopsy which revealed pulmonary arterioles and arteriovenous. The patient had two pregnancies. The symptoms were syncope, attack of dyspnea, and edema. Electrocardiographically a normal. At autopsy there was evidence of marked right ventricular hypertrophy.

Heart sounds were recorded twice at 4 month intervals with identical results. There is a loud S1, the murmur and an early diastolic snap. The murmur may be that of tricuspid regurgitation, there were prominent pulsations in the neck. The early diastolic click is of uncertain origin as Evans (411) has described a similar finding. It is probably a tricuspid opening snap. Both the mitral and the tricuspid valves were normal at autopsy.

## CHAPTER 20

# Pulmonary Arterial Hypertension

The most common cause of pulmonary hypertension is increased back pressure from the left side of the heart from many different causes including systemic arterial hypertension—just as left sided heart failure is the most frequent cause

aspect of cystic fibrosis of the pancreas, pulmonary fibrosis of many causes etc., etc.) Primary pulmonary hypertension is legitimately considered a separate category

Primary pulmonary hypertension occurs with

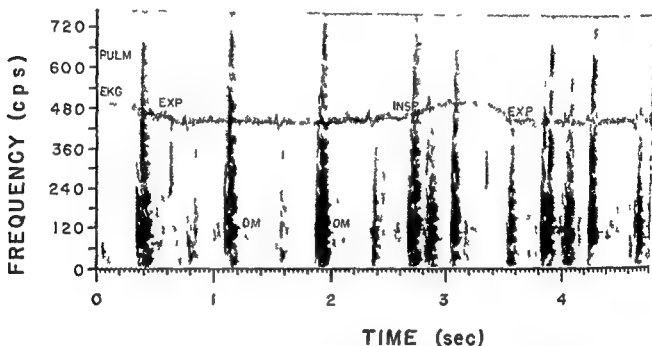
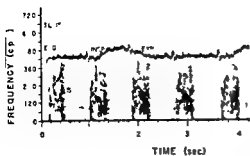


FIG. 437 Multiple pulmonary emboli

Pulmonary area in I S (J55061) 69 year old female with pulmonary hypertension from multiple emboli. Atrial fibrillation S<sub>1</sub> is greatly accentuated with slight splitting especially in inspiration the second (pulmonary) component is much the louder S<sub>2</sub> is almost absent except after a very short diastole. There is a Graham Steell murmur

of right sided heart failure. Multiple pulmonary embolization is an increasingly well recognized cause (1173). Other causes include (1) congenital malformation of the pulmonary vasculature (fetal persistence) usually in combination with septal defect (2) wear and tear from increased pulmonary flow in ASD, VSD and PDA and (3) cor pulmonale due to a variety of types of primary lung disease (e.g. sarcoid, the pulmonary

overwhelming predominance in females and serious symptoms develop usually at about the age of 30 years. Rarely it occurs in children (1584). One of the more attractive suggestions is to the pathogenesis of many of these cases is that thromboplastic material is released into the blood stream and produces tiny fibrin clots which lodge in the lung. The placenta, especially if traumatized is a rich source of thromboplastic material



Concomitantly, merely men trusion is accompanied by release of thrombotic material in some women.

Any pulmonary hypertension which may be present is exaggerated by the presence of high flow in the pulmonary circuit—be it transient as with exercise or permanent as with septal defects.

The pulmonary valve sent a less substantial

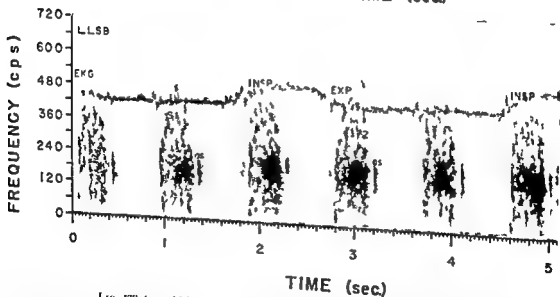
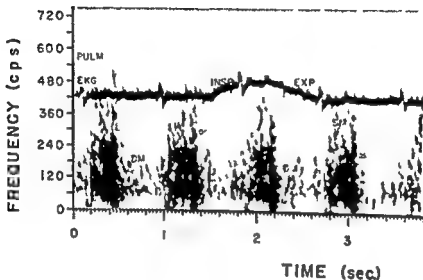


FIGURE 3. Unlike opening snap in primary pulmonary hypertension

A 30-year-old white female was thought clinically to have primary pulmonary hypertension. The clinical conclusion was confirmed at autopsy which revealed pulmonary arterio sclerosis and arterioleclerosis. The patient had had two regurgitations. The symptoms were syncopal attacks, chest heget, megaly and edema. Electrocardiographically confirmed by autopsy there were evidences of marked right ventricular hypertrophy. Heart sound were recorded twice at 4 month intervals with identical result. There is a high systolic murmur and an early diastolic snap. The murmur may be that of tricuspid regurgitation, there were prominent pulsations in the neck. The early diastolic click is of uncertain origin. I have (44) have described a similar finding. It is probably a tricuspid opening snap. But the mitral and the tricuspid valve were normal at autopsy.

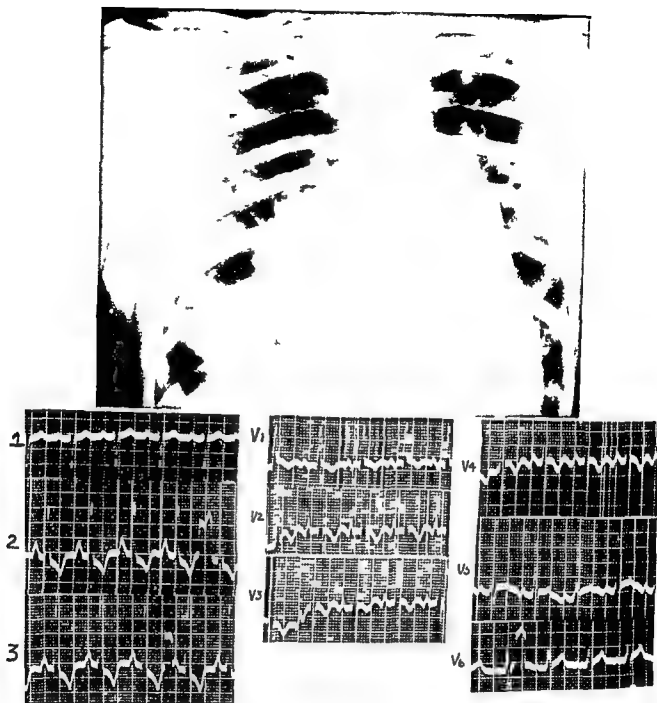


FIG. 439 Functional tricuspid stenosis with pulmonary hypertension (See text.)

than its aortic counterpart with the result that regurgitation with pulmonary hypertension is more likely to occur.

**CARDIOVASCULAR SOUND.** Many of the features are the same as for systemic arterial hypertension, e.g., presystolic gallop. The pulmonary second sound is accentuated and many writers claim that it is a rule is closely split. An early pulmonary systolic click is heard much more regularly than in aortic hypertension, probably be-

cause the pulmonary artery is more superficially located. The early systolic click is usually followed by a systolic murmur generated in the dilated pulmonary artery.

The Graham Steell murmur (Fig. 437) was originally described as the murmur of high pressure in the pulmonary artery of whatever cause and not as an accompaniment of mitral stenosis alone. With severe systemic arterial hypertension of long standing, I have observed (W.A. 599691)

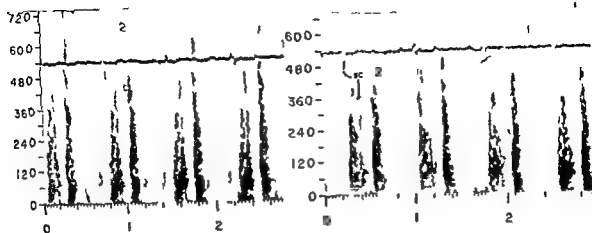


FIG 440 Primary pulmonary hypertension

(Left) Pulmonary area (Right) Apex  $S_2$  greatly accentuated. Early  $S_2$  (chick) loud in 1  $S_2$  split at apex (right). In this 6 year-old male (H.K. 314501) pulmonary arterial pressure was 97/3 mm Hg with a mean value of 55 mm. The pulmonary artery was markedly dilated.

Note: (1)  $S_2$  is accentuated in intensity and frequency, but is not split. (2) In the pulmonary area an early  $S_2$  (chick) was under tandemly misinterpreted as a split  $S_2$ . It is mainly the treble pitch of the sound which is heard in this area. (3) At the apex  $S_2$  is truly split and the early  $S_2$  (chick) is also misinterpreted. (4) An atrial gallop frequent in hypertension of either aortic or pulmonary origin is seen in the pulmonary area. A faint third heart sound is seen in some cycle.

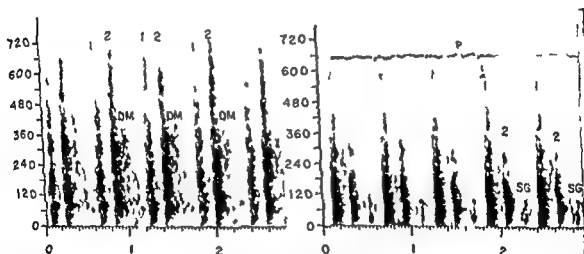


FIG 441 Primary pulmonary hypertension

(Left) Pulmonary area (Right) Apex (reatly intensified) pulmonary chloride sound with murmur of pulmonary regurgitation (left) Summation gallop at apex (right) the IR interval was 0.25 sec. H (681445) 2 year old female had a pulmonary artery pressure of 131/6 mm Hg with a mean of 89.

what was interpreted as a Graham Steell murmur because it was closely confined to the left sternal border was accentuated by inspiration and followed a very loud  $I$ . In general some additional cause such as multiple pulmonary emboli or pulmonary valve stenosis—both difficult to exclude—must be suspected in such case. In one

case (142) of operated cases of mitral stenosis a Graham Steell murmur was found in 6 of 18 patients. It disappeared in all following valvulotomy.

Favans and colleagues (444) heard and recorded what they thought might represent an atrioventricular opening snap in one case of primary



pulmonary hypertension. It was rather late after the second sound and may have represented an unusually snappy third sound. See Figure 438 for a case of possible opening snap from our experience.

In a 17 year old female patient in this hospital (B. D., 745792 ant 26655) who had severe primary pulmonary hypertension, a mid diastolic

rumble was heard at the apex and interpreted as representing the murmur of functional tricuspid stenosis heard in ASD. Autopsy revealed no septal defect, (see Fig. 439). Similar case of functional tricuspid stenosis in association with pulmonary hypertension have been described by a number of writers (see page 327).

## CHAPTER 21

# Miscellaneous Disorders

### DYSRHYTHMIAS

The term *dysrhythmia* is used in preference to *arrhythmia* because some of the conditions discussed are not lacking in rhythm although they are disturbances of the cardiac rhythm e.g. paroxysmal tachycardia.

**SINUS TACHYCARDIA** Tachycardia of any type may be accompanied by accentuation of  $S_1$ . The ventricles are likely to find the AV valves in a wide-open position at the time of contraction. In addition more rapid valve closure results in a snappier first sound with more components of higher frequency.

**ATRIAL TACHYCARDIA** Although as a rule there are no particularly unique features of the heart sound, Figure 442 presents the finding of regular rhythm resulting from the presence of 2:1 block in a case of atrial tachycardia.

**PREMATURE CONTRACTIONS** (844) With any premature contraction whatever the origin the first heart sound may be delayed (61 938 1334) and accentuated because of wide open position of the atrioventricular cusps at the time of ventricular contraction. Schaefer and Little (1334) found that the first sound was more delayed relative to the QRS when it had its origin at the base than when it had its origin at the apex. Most often the PR interval is reduced in cases of atrial premature contractions. Therefore the AV cusps are likely to be widely spread at the time of ventricular contraction according to the considerations reviewed in connection with complete heart block (see p 171). In the case of ventricular premature contraction the AV leaflets may be widely spread merely because ventricular contraction occurs rather early in diastolic filling. In 20 cases Cio (217) found an accentuated  $S_1$  in 6 cases, a diminished  $S_1$  in 7 and in unaltered  $S_1$  in 7.

In ventricular premature contraction both heart sounds are frequently split due to ventricular asynchrony (Fig 443). The QRS is widened in ventricular premature contractions (PVC) just as in bundle branch block. A PVC arising in the left ventricle tends to have the pattern of right bundle branch block and vice versa. For some reason the heart sound especially the first are more regularly split with PVC's than with bundle branch block. Splitting of the heart sound does not occur with atrial premature contractions because there is as a rule no widening of the QRS and no ventricular asynchrony.

In extrasystoles there is usually a direct relation ship between the intensity of  $S_1$  (Fig 442) and the delay of that sound since both phenomena are based on the position of the AV valve at the time of ventricular contraction (302). However when a ventricular extrasystole falls in the rapid filling phase of diastole  $S_1$  may not be accentuated despite a low position of the cusps due to poor filling of the ventricle.

There are several reasons (302) why the first sound is more often intensified and delayed in atrial than in ventricular extrasystoles: (1) Splitting of  $S_1$  in VPC's divides or distributes the intensification. (2) Atrial extrasystoles usually have a short PR interval which is likely to result in a loud  $S_1$ . (3) Ventricular filling may be more complete in atrial extrasystoles because atrial contraction has contributed to ventricular inflow.

The Q-T interval (or the interval between the onset of ventricular systole and the first heart sound) varies with the PR interval (or the interval between atrial and ventricular systole). See Figure 446.

The second heart sound is usually reduced in amplitude or completely absent in extrasystoles (217).

pulmonary hypertension. It was rather late after the second sound and may have represented an unusually snappy third sound. See Figure 438 for a case of possible opening snap from our experience.

In a 17 year old female patient in this hospital (B. D., 747792, aut. 2665a) who had severe primary pulmonary hypertension, a mid diastolic

rumble was heard at the apex and interpreted as representing the murmur of functional tricuspid stenosis heard in ASD. Autopsy revealed no septal defect, (see Fig. 439). Similar cases of functional tricuspid stenosis in association with pulmonary hypertension have been described by a number of writers (see page 327).

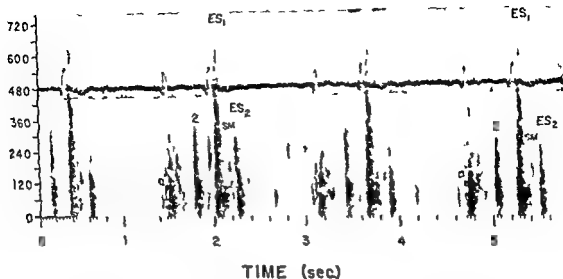
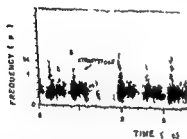


FIG. 413 Bigeminy

Here is presented the recording from the aortic area in a patient (W. C. 15/847) with aortic sclerotic cardiovascular disease and pulmonic bigeminy. Note that the extra systolic first sound is greatly accentuated—probably caused by the wide opening of the AV valves at the time the extra systole occurs. Note secondly that whereas in the normal cycle there is a sound which precedes the Q wave of the I & II leads, the extra systole occurs before the first sound of the ventricular extra systole. The atrium is of course inactive in the case of ventricular extra systoles unless retrograde conduction occurs. In the third place, note that the second sound usually diminishes in the extra systole as compared with the normal main beat. This is almost certainly related to low stroke output and low aortic (and pulmonary) diastolic pressure with the extra systole. Finally, note the considerable abbreviation of S<sub>1</sub> in the case of the premature beat. S<sub>1</sub> is shortened simply because of delay in the onset of the first sound. This delay is probably on the same basis as that seen with atrial fibrillation and is related in some way to the absence of preceding atrial contraction. The wide open position of the AV valves may demand a longer time for closure. Note the 60 cycle electrical interference. Altogether there are four or five short sounds (cracks and crackles) which are easily identified as artifacts.

FIG. 414 Effect of extra systole on murmurs of S<sub>1</sub> at 1 M.

In W. W. (100033) 26 year old male at the aortic area the Christy murmur with the extra systole is small but after the compensatory pause it is larger than normal. The changes in the diastolic murmur are limited.

Changing intensity of S<sub>1</sub> is a valuable diagnostic sign in atrial flutter. Variability of S<sub>1</sub> is not always present but when it is covered seems to have it but is in variable relation of atrial to ventricular systole with variability in the position of

the AV valves at the onset of ventricular systole. In the electrocardiogram in flutter one observes fairly frequently beat to beat variation in the relation of the QRS to the saw tooth flutter waves. Libby and colleagues (841) describe a variable triple rhythm in atrial flutter.

In atrial fibrillation there is usually beat to beat variability in both the intensity of the first sound and its degree of lag after the QRS of the I & II (1113). This variability is secondary to the variability in the point in diastolic filling at which ventricular contraction occurs and corresponding variability in the position of the AV valves at that time (36 37 1247 1336) (see figure 447). When mitral stenosis is combined with atrial fibrillation the variability in S<sub>1</sub> intensity is exaggerated (980). The high atrial pressure is responsible for delay in the first sound. On the length of the preceding diastole depends how much the

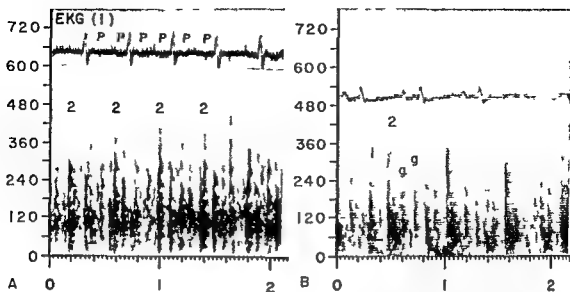


FIG. 442. Quadruple rhythm.

In the example presented in (left) (the aortic area) the 65 year old patient (C. W. 252981) had paroxysmal atrial tachycardia with 2:1 atrioventricular block. Each atrial contraction was accompanied by a sound—one in systole one in diastole. The second heart sound was slightly split in some instances. The occurrence of an atrial sound in ventricular systole when the atrioventricular valves are certainly closed is evidence that tensing of the atrium and not movement of blood into the ventricle was primarily responsible for its production. In the electrocardiogram shown here the presence of two negative P waves between each two QRS is not clearly demonstrated but was clear from conventional electrocardiograms.

In the recording at right (also from the aortic area and presented here by way of contrast) the quadruple rhythm results from the presence of both protodiastolic and presystolic gallop (g) sounds. The patient has cor pulmonale caused by multiple pulmonary emboli. The second sound is accentuated and is followed by a short diastolic murmur of presumed pulmonary origin. Pulmonary is demonstrated by the electrocardiogram. An early systolic click of dilated pulmonary artery makes this a quintuple rhythm in some cycles.

A short early systolic murmur of low intensity may occur with ventricular premature contractions even though not present with normal beats. The basis is not clear. Atrioventricular regurgitation—which Little (956) shows does occur with extrasystoles before the AV valves are closed—cannot account for it since it is occurring after closure of the AV valves as indicated by the first sound. Systolic murmurs of either the regurgitant or the ejection type are attenuated with the extrasystole (Figs. 444 and 445).

The heart sounds in the cycle after the compensatory pause show differences from those in the average normal cycles. The differences are the result mainly of the increased stroke volume which in turn results from the long diastolic filling phase. The first sound is accentuated. The second sound is also likely to be accentuated because of moderately increased pressure in both circuits. Murmurs tend to be exaggerated in the post-pause cycle (Fig. 444). Alternation in the intensity

of the heart sounds may parallel the pulsus alternans which may occur in the several beats following an extrasystole (217).

**ATRIAL FLUTTER.** As a rule atrial sounds are audible only when complete atrioventricular dissociation is present. With the combination of atrial flutter and third degree heart block, atrial sounds are almost invariably present as indicated by the relatively large number of reports (87, 101, 215, 941, 1317). Alternation in the amplitude of the atrial sounds related presumably to alternation in the strength of atrial contraction has been described (87). Hecht and Myers (660) found very loud atrial sounds in two cases of atrial flutter with AV dissociation. The sounds were loudest in the pulmonic and aortic areas. Interestingly they occurred during ventricular systole as well as diastole. The authors raised the question of whether they might in fact represent atrial pleuropericardial clicks.

Harvey and Levine (655) have emphasized

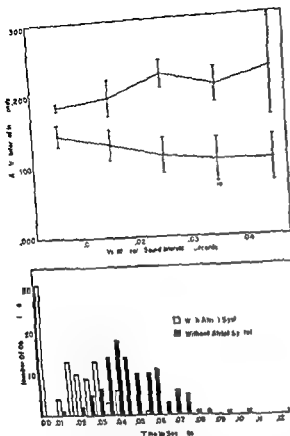


FIG. 448 (above) Relation between interval from onset of ventricular systole to the first sound ( $a_1$ ) and interval from  $a_1$  to ventricular systole ( $a_1-v$ ). (right) Ventricular extra systoles were induced by artificial means in dog. (Below) Ventricular extra systoles occurring without a preceding atrial contraction displayed a longer delay between the onset of ventricular systole and the first sound ( $a_1$ ) than did ventricular extra systoles which occurred after an atrial systole. (From Little, Hilton and Schaefer (245).)

when  $S_1$  occurred in early diastole rather than being accentuated as in subjects with atrial fibrillation and normal AV valve (Fig. 449). Rydberg proposed a diagnostic test based on this observation for use in cases of atrial fibrillation when the presence of mitral stenosis is uncertain. Ray and Berkhof (1247) extended the observation. They agreed that although a majority of cases of mitral stenosis (6 out of 10 in their study) and atrial fibrillation show no variation in the intensity of  $S_1$ , there are other patients (4 out of 10) who do show variation which is however of strikingly different pattern from that in cases of atrial fibrillation without mitral stenosis. The intensity

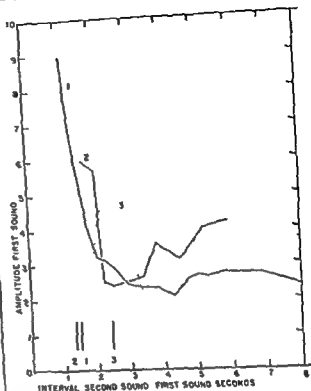


FIG. 449 Variation in  $S_1$  with  $S_1-S_2$  interval in atrial fibrillation with mitral stenosis. (Three cases.) The lines indicate the timing of the periodic gallop in each case. (From Livland (1238).)

however, does decline with increasing length of preceding diastole reaching a minimum only at 0.5 to 0.7 sec values for the diastolic period. The observers concluded that the patients with this phenomenon had phantoms and low diastolic filling. The question of the influence of cycle length on  $S_1$  in cases of mitral stenosis and atrial fibrillation is complicated by the necessity to consider the mitral and tricuspid components separately (7233).

Meda and Smetana (1090) in 31 cases of atrial fibrillation in patients with various forms of heart disease (rheumatic mitral disease, hypertension, arteriosclerotic heart disease) could find no relation between the Q-T interval and the duration of the preceding diastole (Fig. 449). Intensity of  $S_1$  showed a direct or inverse linear or hyperbolic relationship to the duration of the previous diastolic period—or no relation at all. The clinical diagnosis was unimportant in determining which relation held. It did appear that in ranges of lower ventricular rates  $S_1$  became

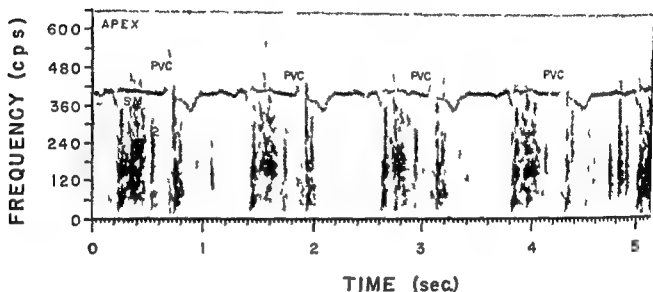
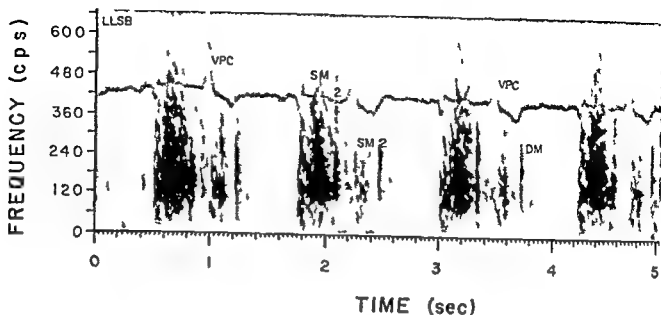


FIG. 445 Bigeminy of digitalis intoxication

I. A. (562915) 54 years old had a history of syphilis treated eight years previously with penicillin. The systolic murmur at the base was transmitted into the neck and accompanied by thrill. Fluorocopy showed no dilatation of the aorta. LLSB (A) and apex (B). The systolic murmur has the pattern typical of aortic stenosis which in this case was thought to have been only relative (p. 264). S<sub>1</sub> is followed by a decrescendo diastolic murmur which is interrupted by an extrasystole. Very little murmur accompanies the extrasystole. In apex both heart sounds especially the first are split with the extrasystole.

atrium is decompressed by the time ventricular contraction occurs. The longer diastole is the lower is the level to which atrial pressure falls, and the less the delay of the first sound. With shorter preceding diastolic periods the first sound might be expected to be louder (as well as more delayed) because the valve curtain is belted further toward the ventricle. Presumably because of a floating up of the valve leaflets as a result of ventricular fill

ing, a semiclosed position is attained and less noise produced during closure. In patients with normal mitral valves, minimal amplitude of S<sub>1</sub> occurred with diastolic periods between 0.20 and 0.25 sec (Fig. 447). Thereafter there was at times a secondary increase in amplitude. However, Rydman (40) found that relatively little variation in the intensity of S<sub>1</sub> occurred in patients with MS and that if anything intensity was less

In conclusion it should be pointed out that in addition to the particular changes in the heart sound noted above many of which can be detected by stethoscopy in cultivation the most convenient bedside method for gauging the effect of carotid massage compression in cases of tachycardia is a large portion of Levine and Hare's *Clinical Cultivation of the Heart* (1949) was devoted to the cultivatory features of arrhythmia. To the authors is owed a general understanding of the distinctive behavior of each type of tachycardia (96) when pressure is applied to a carotid sinus artery and then released.

**Second tachycardia**—gradual slowing and gradual return to original rate.

**Paroxysmal atrial tachycardia**—abrupt cessation of heart beat followed by normal rhythm or abrupt resumption of previous rate.

**Atrial flutter**—slowing and return in jerky manner because of varying atrioventricular block.

**Atrial fibrillation**—light effects at the most.

**Ventricular tachycardia**—no effect.

### CONDUCTION DEFECTS

With **first degree atrioventricular block** manifested in the EKG by prolongation of the PR interval the first sound is likely to be muffled because of the relatively high position of the atrioventricular valve curtain at the time of ventricular contraction (p. 171). However with the abnormally short PR interval of the Wolff-Parkinson-White syndrome Levine and Hare (1949, p. 15) failed to find that the first sound is louder and sharper. On the other hand in the syndrome of short PR interval with normal QRS complex and susceptibility to paroxysmal tachycardia Lowy, Cronson and Levine (1947) did note a snapping A<sub>1</sub>.

In **second degree heart block** in which only part of the atrial impulse is conducted to the ventricle there may be no particular cultivatory change. If there is Wenckebach phenomenon—progressive lengthening of the PR interval and final failure of conduction—the intensity of the first sound may show a progressive diminution. In 2:1 or 3:1 heart block with a slow ventricular rate (in the 40's) there may be an early diastolic murmur of the Carey-Coombs type. This murmur may occur merely with very low ventricular

rate but in the situation mentioned it is likely that the occurrence of atrial systole at the same time as rapid ventricular filling on a passive basis contributes to the murmur. If organic mitral stenosis is present with 2:1 block there may be two aortic valve murmurs in diastole (278).

In **third degree heart block**—complete atrioventricular dissociation—there are cycles of cycle variation in the intensity of the first heart sound (76, 274, 378, 418) as well as aortic murmur and diastolic murmur of note. Atrial heart sound may also be heard and in most cases of AV dissociation of some degree some murmur is to be maximally audible at theortic area and down the right sternal border in general. *Systolic murmur* is an expression for the sound the origin of the term is obvious.

When the PR interval is appropriately short the first sound may be very loud the so-called *bruit de canon*. In young subjects there is likely to be intensification of S<sub>1</sub> with short PR interval (0.14-0.20 sec.) and with PR interval in excess of 0.32 sec. (fig. 4-3). In older subjects the second zone of intensification may not occur (131, 152, 178). Children (fig. 4-4) resemble therefore the millman's wheel the case in which Durr (111) studied mitral valve motion (fig. 4-5) and the dog in which Boyer (see fig. 129) studied S<sub>1</sub> intensity in artificially induced heart block. On the basis of a single case Vane and Lill (1489) claimed that when rheumatic mitral valve disease is combined with third degree block accentuation of the first sound may occur only with longer PR interval of the order of 0.31 sec.

The variability in the first heart sound is related to the variability in the relationship of ventricular to atrial systole. Specifically there is every reason to believe that the variation in S<sub>1</sub> intensity is due to variation in the position of the AV valves at the time of ventricular systole. An older theory dates at least from 1912. Griffith of Manchester (197) an early student of the phenomenon held that the accentuation of the first sound was caused by a coincidence with summation of atrial and ventricular systole. This theory would not explain the accentuation with longer PR interval and is on the whole untenable.

Laubry and Puddu (530) found that *bruit de canon* can occur with third degree heart block and a rapid ventricular rate. This is not surpris-



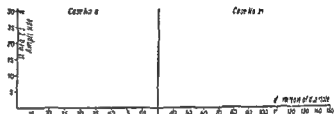
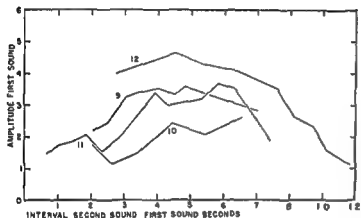
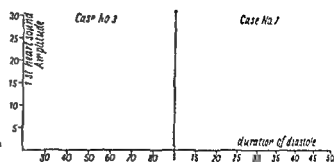
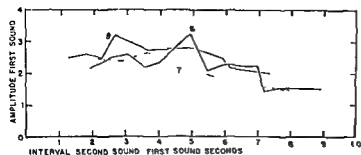


FIG. 448. Amplitude of  $S_1$  in atrial fibrillation with mitral stenosis.

Note the relatively minor variability in the amplitude of  $S_1$ . If anything the intensity is less with shorter diastolic period. (From Ryd and (1936).)

louder when diastole was longer, whereas at very rapid rates the converse relationship appeared to obtain.

There is likely to be a short early systolic murmur of low intensity. This murmur may vary from cycle to cycle.

Variability is the rule in atrial fibrillation and the second sound diastolic murmurs and the mitral opening snap show it too. The second sound may be faint or absent if ventricular contraction occurs so early in the preceding diastole that the atrial valves are lifted little or not at all. Sometimes a paradox results. The first sound is accentuated and the second sound is diminished or lost. In tricuspid or mitral stenosis the opening snap and/or the onset of the diastolic murmur are closer to the second sound when the preceding diastole is short. The intensity of the murmur of aortic or pulmonary regurgitation is greater in the cycle following a long diastolic period because of greater cardiac output and higher pressure at the onset of diastole.

FIG. 449. Relation of  $S_1$  intensity to duration of preceding diastole.

The two variables were significantly correlated in only 7 of 23 cases. The direct relationship illustrated above by case 3 is an instance of decompressed mitral stenosis, mean ventricular rate 73 per min. The indirect relationship is illustrated above by case 7, in which there was myocardial infarction, mean ventricular rate 103 per min. No relationship exists in case 5, in which there was no other evidence of heart disease. A biphasic relationship was found in case 21, in which there was combined mitral and aortic valvular disease. (From Medt and Seinfeldt (1950).)

Some (1062) have claimed an increased incidence of systolic murmur in association with atrial fibrillation as well as with ventricular extrasystoles and pointed to the observations of Diley, McMillan and Gorlin (323). The absence of atrial systole at the usual time just before ventricular systole might explain regurgitation before closure of the atrioventricular valves, but it is difficult to see how regurgitation—and murmur—could occur on this basis after the first sound.

In *ventricular tachycardia* (856) variability in the intensity of the heart sounds is a characteristic feature. Variability in the intensity of  $S_1$  has its basis in a varying relation of atrial to ventricular systole. The presence of this variability requires that the atria be beating independently of the ventricles, under control of the sinoatrial node or other supraventricular pacemaker. Slight variability in rate is also a clue to the diagnosis. The heart sounds are often so rapid and one either the first or the second so faint that there is risk of counting the rate at just half the correct one.

## MISCELLANEOUS DISORDERS

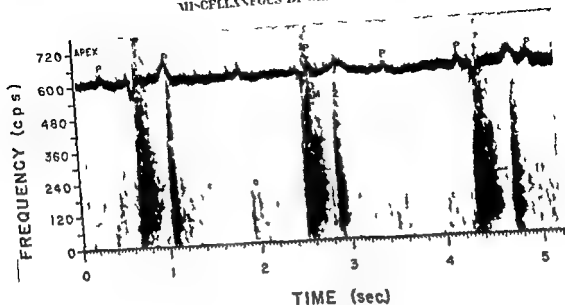
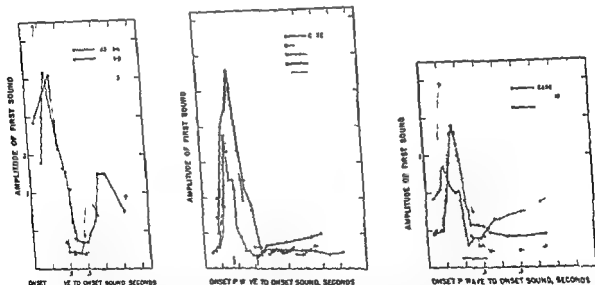


FIG. 452. Complete heart block.

H. J. (1568) 60 year old man had Adam Stokes disease. There is an ejection type of systolic murmur probably caused by the large stroke volume. Atrial sounds are faintly seen. The one marked (a) seems to be doublet.

FIG. 453. Variation of mean peak amplitude of  $S_1$  against time of onset of  $S_1$  after P wave.

(Left) Four children. (Middle) Five adults in whom doublet atrial sounds were recorded: the two vertical lines near the bottom represent mean timing of the two components from onset of the first wave. (Right) Four adults with atrial murmur at apex: the horizontal line near the bottom represents the mean timing of the murmur from onset of the first wave. (From Ryland (1934).)

cupps bring them in closer apposition with smaller excursion in production of the first heart sound.

A systolic murmur has its basis in the large stroke volume which is necessarily present in heart block (Fig. 452). The murmur has the char-

acteristics of an ejection systolic murmur as outlined by Leatham (836). Specifically the murmur does not extend throughout systole but rather is separated from the second sound by a brief interval. The second sound in complete heart block may be split if the idioventricular focus is situated in

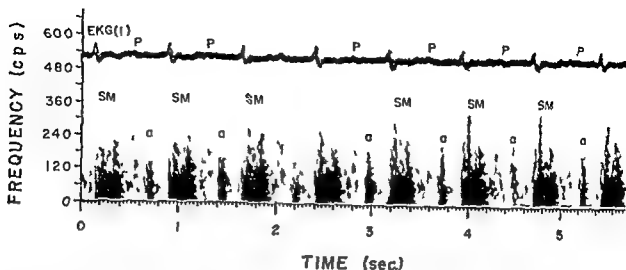


FIG. 450 Prolonged PR interval in rheumatic heart disease

Apex in A I (299576) in whom the predominant lesions were aortic regurgitation and mitral regurgitation. The relatively faint early diastolic murmur is transmitted from the base. An atrial sound (or short murmur) is displaced into mid diastole by the long PR interval.

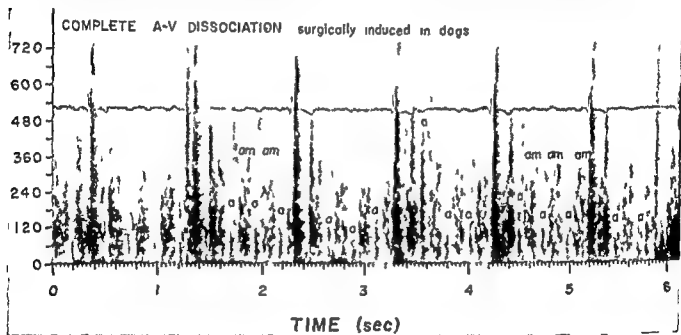


FIG. 451 Surgically induced complete heart block in the dog

Third degree heart block was produced by Dr T. I. Sturzl by the method he and his collaborators have described elsewhere. A circumscribed sound (a) occurs with tensing of the atria and is followed by a murmur (am) related presumably to the passage of blood into the ventricle. The occurrence of this phenomenon—an atrial sound followed by an atrial murmur—has been described in elderly patients with complete heart block (1335). Variation in the intensity and frequency position of the first sound is demonstrated. This record is too short to demonstrate convincingly that the louder sounds occur with the shorter PR intervals but such did appear to be the case. The recording was made one month after operation for creation of heart block. Although it seems unlikely a pericardial origin of the murmur following each atrial sound cannot be unequivocally excluded.

inasmuch as variability of  $S_1$  is seen with ventricular tachycardia, if there is a regular atrial beat (see p. 438).

Stead and Kunkel (1435) observed that in

cases in which it was possible to separate the effects of PR duration from that of the duration of diastole,  $S_1$  became fainter after longer diastolic periods. Presumably, floating up of the AV

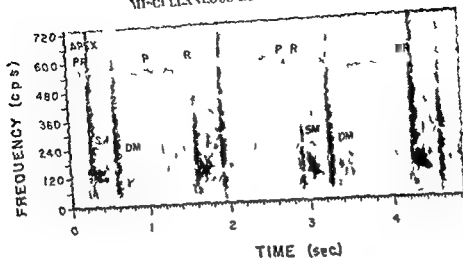


FIG. 4b. Congenital complete heart block.

ApeX in V V (B180 f) a year old white male who has complete atrioventricular dissociation as an isolated congenital anomaly. Note (1) variability in the intensity of  $S_1$  depending on the preceding R interval (2) the systolic murmur with the spectral characteristic of the Still murmur (3) the third heart sound which appears to be followed by a short rumble.

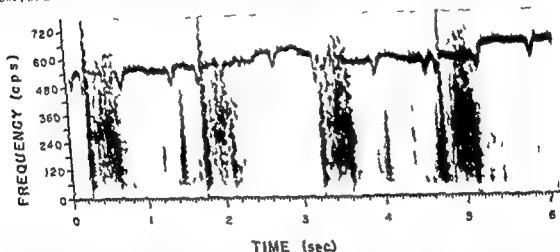


FIG. 4c. Pulmonary area in B C (44131) 14 year old girl with pulmonary stenosis and congenital atrioventricular dissociation.  $S_1$  systolic pressure in the right ventricle was 180 mm Hg. An atrial septal defect was also noted on cardiac catheterization. Before operation an infundibular location of the pulmonary stenosis was considered likely because of the absence of post-stenotic dilatation of the pulmonary artery. On the day following the above recording the extent of the infundibular pulmonary stenosis was more fully performed and rehypothermia. The rising femoral diastolic pressure in the intensity of  $S_1$  a systolic murmur which extends over the second and third aortic closure sounds and middle atrial heart sound. An early diastolic murmur was present at the apex.

and about 0.20-0.21 sec after the onset of the first wave. If their interpretation that the wave is produced by momentary tricuspid closure is correct explanation for the atrial sound in these cases may be provided. The Henderson-Johnson mechanism—drawing in of the cup in the wake of the ejection jet—may be involved in AV valve closure in such cases.

Froment and colleagues (459) in writing on the atrial sound in heart block (called galop du bloc by Louis Callard in 1911) point out that the atrial sound may be single or double depending on the position of atrial systole in ventricular

<sup>1</sup> Many workers (903) (315) have noted that the atrial sound is frequently double in complete heart block.

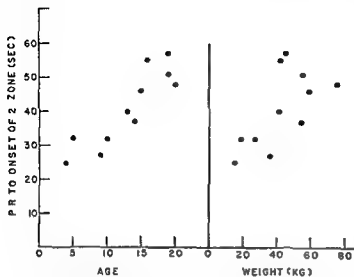


FIG 454 The relationship between onset of secondary zone of  $S_1$  accentuation and age and weight in complete heart block in man (From Sherrin Tarr and Rydand (1952))

the ventricles at a site such that prolongation of the QRS results. If the intraventricular focus is high—close to the AV node—prolongation of the QRS does not occur and splitting of  $S_1$  is not to be expected. With very slow ventricular rates an early diastolic murmur of the Carey Coombs type may occur on the basis of large AV valve flow and dilatation of the ventricle. Wood (1590 p. 324) states that it was present in over  $\frac{3}{4}$  of the present series [of congenital complete heart block], particularly when the rate was under 50.

Paul and co-workers (1187) found such a murmur in all six of the patients they studied and attributed it to high intraventricular flow accelerated by recurring atrial systole.

A murmur in ventricular diastole following atrial contraction has been described by Rydand (1335) in elderly subjects. In these cases there was often an atrial heart sound followed after a brief interval by a blubbery murmur, the Rydand murmur. Rydand referred to this as an atriodiastolic murmur. The murmur had its onset after the onset of the P wave by an interval of 0.11 to 0.23, usually about 0.16 sec. Windholz and Grayson (1573) in describing intrusion of the aortic root into the mitral orifice in hypertensive disease described three patients in whom this diagnosis was possible in life because of calcification of the fibrous skeleton of the heart. All three had a diastolic murmur. Two had heart block and were the same patients as those reported

by Rydand. It is possible that calcification of the annulus fibrous mitralis and/or intrusion of the aortic root is responsible for the Rydand murmur. However, Rydand's own explanation (1335) seems more likely. "After auricular systole, normal mitral leaflets are floated nearly together. In the aged, they remain longer and more fixed in this position because of their increased rigidity. The murmur occurs then with continued forward flow through the relatively narrow orifice. It is loudest in the part of early (ventricular) diastole which follows the phase of rapid filling." Figure 451 shows both atrial heart sounds and the Rydand murmur in a dog with surgically induced complete heart block.

Atrial heart sounds are heard in possibly 50 per cent of cases of complete heart block with the atrial pacemaker in the SA node. Atrial heart sounds at a rapid rate are heard as a rule in case of complete heart block with atrial flutter and infrequent syndrome. In cases of heart block Flach and Heeger (435) have pointed out a small sharp wave after the a wave in the jugular venous pulse.

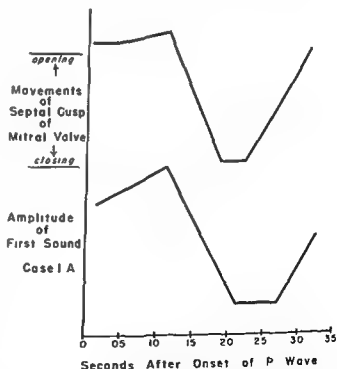


FIG 455 Curve (below) relating amplitude of first sound in complete heart block to time after onset of P wave (from Sherrin Tarr and Rydand (1332)) and for comparison curve (above) of movement of anterior leaflet of mitral valve in perfused heart of cat (after Dean (341))

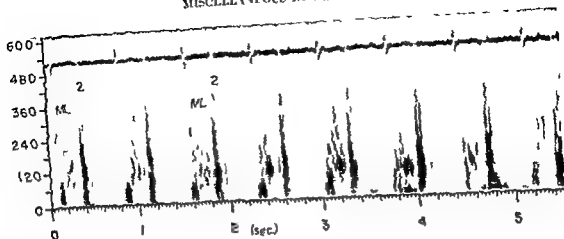


FIG 4-3 Mean Lerman scratch of toxic patient

This recording was made at the third left intercostal space in a patient with severe hyperthyroidism. A scratchy systolic murmur was present. In order to display better the scratchy quality of the systolic murmur the analysis were made with filter system 1 rather than the cut tomograph filter system C (Fig. 4-3). The systolic murmur ended before the end of systole. Its mechanism was thought to be dilatation of the pulmonary artery with flow through it increased both in volume and velocity. This is an ejection systolic murmur which stops slightly before the next cardiac cycle. The second sound is split in some cardiac cycles.

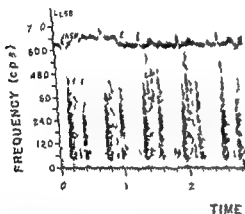


FIG 4-4 Lerman Means scratch

LLSB in L. D. (37 years old) 46 years old with severe thyrotoxicosis. As in the case of pericardial friction rubs it is difficult to identify the physical basis of the scratchy quality. However as in pericardial friction rubs it is a rather diffuse frequency pattern and discrete transients can not be identified. The murmur is not holosystolic.

of blood flow which contributes to the generation of the murmur is reflected by the reduced circulation time. Blumgart and colleagues (124) found that with a large increase in basal metabolic rate of 33 per cent the mean velocity of blood flow through the lung was increased by 83 per cent. The increase in velocity of blood flow is out

of proportion to that which accompanies a corresponding increase in oxygen consumption from work in normal subjects (144). The duration of ventricular systole indicated in the phonocardiogram by the interval between the first and second sound is reduced more than would be predicted on the basis of tachycardia alone. Contraction of the ventricle to accommodate by the room appears to be the basis. Wood (190 p. 880) states that a Carey-Coombs murmur may occur in thyrotoxicosis on the basis of hyperdynamic atrioventricular flow. On the other hand Löffler and Lerman (147) state that they never heard a diastolic murmur in adult patients. One might expect to find it especially in children and adolescents with thyrotoxicosis since these age groups seem most prone to functional mitral stenosis. With this in mind I reviewed the cases of childhood thyrotoxicosis in the Johns Hopkins. Ilo put it and found no case in which a diastolic murmur was described.

The thyroid bruit shows an accentuation in systole as a result of its basis in what is functionally an arteriovenous fistula. Venous hum in the neck is frequent in thyrotoxicosis but shows maximal intensity and frequency span in diastole after opening of the tricuspid valve. A continuous thyroid bruit occurs in about 20 per cent of

diastole. In the patient they studied it was likely to be double when the EKG indicated a 1 P interval of 0.06 sec. and likely to be single with longer or shorter intervals. The second element was usually loudest and was the one present when the gallop was single. Yet it was the first element which seemed to correspond to the usual presystolic gallop since the second element occurred about 0.20 sec. after the beginning of the P wave rather than the 0.15 sec. usual for the presystolic gallop.

Steid and Kunkel (1435) and Wolferth and Margolies (1578) noted that the atrial sound (or auricular murmur, as they termed it) appears to vary with atrioventricular pressure difference. Early in diastole this difference is maximal and the murmur loud, in the latter portion of diastole the difference is much less and the murmur either faint or absent. The observation leads to the conclusion that the atrial sound in question is produced by (or in response to) the flow of blood into the ventricle and not by contraction of the atrium *per se*. Bramwell (151) made similar observations on a variable intensity of the atrial sound in a patient with complete heart block who was, however, thought by the author to have mitral stenosis, also.

The auscultatory changes in bundle branch block are of note particularly in regard to the splitting of the second heart sound as outlined on pp. 159 to 167.

Braunwald and his colleagues (164, 165, 168) found that, in 15 patients with right bundle branch block and evidences of right ventricular hypertrophy, the onset of right ventricular contraction (as indicated by ventricular pressure curves) followed the onset of the QRS complex by normal intervals (0.045–0.075 sec.) indicating that significant right ventricular conduction disturbance did not exist. On the other hand in six subjects with right bundle branch block but without heart disease the electro-mechanical interval was prolonged (0.095–0.110 sec.) confirming the presence of conduction disturbance. In congenital bundle branch block splitting of the first heart sound is frequently found, whereas it is unusual in acquired forms of bundle branch block, facts consistent with Braunwald's findings.

Contro and Lundin (286) have emphasized the rarity of splitting of  $S_1$  in bundle branch block although splitting of  $S$  is frequent. They did find prolongation of the first sound in all of the 24 cases they studied. Therefore they concluded that the first sound is normally too long a sound with much overlapping of its components, for one to expect separation of the components in bundle branch block.

Earlier stethoscopic studies (794, 795) claimed a high incidence of splitting of  $S_1$  in bundle branch block. A presystolic gallop occurs rather frequently with bundle branch block, because of the underlying heart disease, and may create an illusion of splitting of the first sound (779).

With the Wolff-Parkinson-White syndrome splitting of  $S$  is less consistently present than in bundle branch block (1374–1419).

### HYPERTOXICOSIS

The auscultatory findings in the heart in hyperthyroidism may contribute to simulation of other conditions, particularly mitral stenosis. The first sound at the apex is often snappy which, with dilation of the pulmonary artery, straightening of the left heart border on x-ray (Fig. 461), enlargement of the left atrium, atrial fibrillation accentuated P and systolic murmur may lead to this mistaken diagnosis (947). (The PR interval of the electrocardiogram is sometimes prolonged.) Quicker contraction of the ventricle is probably responsible (p. 169) for the sharp first sound, the basis of this change is probably the interplay between thyroid hormone and adrenaline which is circulating or produced locally at the sympathetic nerve endings. The scratchy systolic murmur at the left sternal border (Figs. 458 to 460)—the German Meins scratch—can easily suggest pericarditis (1450). This murmur appears to be produced by high flow in the pulmonary artery. Dilation of the pulmonary artery brings the site of murmur production to a superficial level (accounting perhaps for its superficial quality). (A different mechanism—in effect that it is indeed a rub—was proposed in 1920 by Goodall (567) who wrote as follows: "A superficial pericardial rub is often heard; this is most common over the pulmonary base. It is probably produced mechanically.") The increased velocity





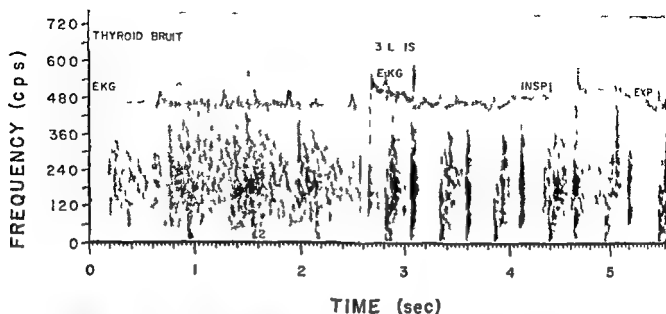


FIG. 460 Thyroid bruit and Iermin Means scratch. F. W. (69137) 33 year old female had typical Graves disease.



FIG. 461 Dilated pulmonary artery in a 28 year old woman with thyrotoxicosis. Fluoroscopically there was an increased amplitude of border pulsations. (Courtesy of Cooley and Sloan (291).)

cases a bruit limited to systole is present in the majority of the rest (929).

The nature of thyroid bruit is that of an AV fistula is well corroborated by the finding of a high degree of oxygen saturation (92 per cent) in

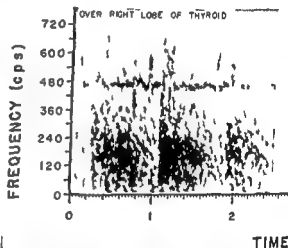


FIG. 462 Persistence of thyroid bruit long after restoration of euthyroid state with thiourea compound. J. H. (271395) a female patient born in 1938 was first admitted in April 1956 with typical Graves disease. A loud thyroid bruit was present. Laboratory confirmation of the diagnosis included cholesterol 112 mg. per cent, BMR + 64 per cent, butanol extractable iodine 18.2  $\mu$ g. per 100 cc. serum. She was treated with Thiopazole. One year later—April 1957—when the record here was made the patient was euthyroid. The protein bound iodine had fallen to 4.0  $\mu$ g. per 100 cc. serum. Despite this the goiter and the bruit persisted. The record shows this in striking form.

the thyroid vein of thyrotoxic patients (1952). At operation in past decades when patients were not so satisfactorily prepared for surgery, it was now the surgeon often found large veins which if cut squirted arterial blood rhythmically like arteries. As with other murmurs of the arteriovenous type the thyroid bruit may be musical. The

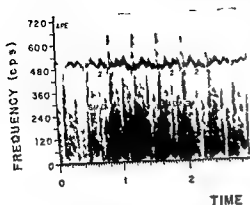


FIG 464 Carey Coombs murmur in anemia

Aperin J C (B'4063) aged 30 months who suffered from severe nutritional (iron deficiency) anemia with hematocrit of 13 per cent hemoglobin of 2.3 gm per cent. There is a decrescendo systolic murmur which tops lightly before the second sound. Because of the rapid rate and short diastolic murmur is short. However there is no question but that this is a murmur and not a gallop.

A 3-year-old woman with severe pernicious anemia hemoglobin only 8 per cent of normal and marked cardiac enlargement. An aortic diastolic murmur disappeared with improvement in the anemia. There are a few other similar reports (494-513 888 p 207 1574). Sometimes a venous hum transmitted over the upper part of the anterior chest may suggest such a murmur when the murmur is usually continuous. A venous murmur which is largely limited to diastole does not begin with the second sound but after a brief interval at a point corresponding to the opening of the tricuspid valve. Occasionally at times the dilatation of the right ventricular outflow tract and pulmonary artery is sufficient to result in pulmonary regurgitation as was suggested by Gallavardin in 1908 (513). The presence in anemia of wide pulse pressure, pistol foot sounds and capillary pulsations increases the confusion with aortic regurgitation.

#### RHEUMATIC CARDITIS

In the pancarditis of acute rheumatic fever (ARF) auricular changes attributable to valvular myocardial and pericardial involvement occur as well as changes associated with conduction defects and arrhythmias.

The most frequent valvular change is one pro-

ducing in apical systolic murmur which may have a partially musical quality (Fig 465). Lurie (988) emphasizes the frequent presence of a harsher musical pulmonary systolic murmur which often has a diamond shape in the phonocardiogram. What its pathogenesis may be and what its diastolic function if any is from the functional murmur described by Still (p 244) are uncertain. Because of the high frequency of functional systolic murmurs in the same age group as that affected by rheumatic fever, Lurie and Bridgen (883) insisted that only a pure systolic murmur (or a diastolic murmur) can be taken as indication of valvulitis in acute rheumatic fever.

The Carey Coombs murmur, an early diastolic murmur usually following an accentuated third heart sound may be present in the early stages of ARF without permanent residual of mitral stenosis (40 97 113 115 1604). Wood (190) states that a Carey Coombs murmur is present in 75 to 80 per cent of cases of rheumatic carditis. It is not always easy to be certain, especially or oculo-graphically whether one is dealing with a third heart sound or a short murmur. The distinction can usually be made with the spectral PCG which usually will show in such cases either a circumcribed sound or a circumcribed sound followed by a short murmur. The juxtaposition of third and fourth heart sounds may suggest a short murmur (1459). Friedman and Harris (485) found a diastolic murmur in 88 of 115 cases. A murmur of aortic regurgitation does not develop as early as a rule and once it has appeared it rarely disappears completely. Wood (190 p 298) refers to its disappearance. In one of my patients (P I 219593) evidence of profound aortic regurgitation was already present at the age of four or five years. In the next four or five years signs of aortic stenosis progressed under observation as the aortic stenosis progressed the aortic regurgitation regressed to the point that no diastolic murmur was audible. At death at 25 years there was necropsy evidence of trivalvular (aortic mitral tricuspid) stenosis. The above sequence of events is an exception to the rule that once developed the murmur of aortic regurgitation persists. Glizebrook (557A) described two patients in whom a murmur of aortic regurgitation appeared 20 days after the onset of acute rheumatic fever and dis-

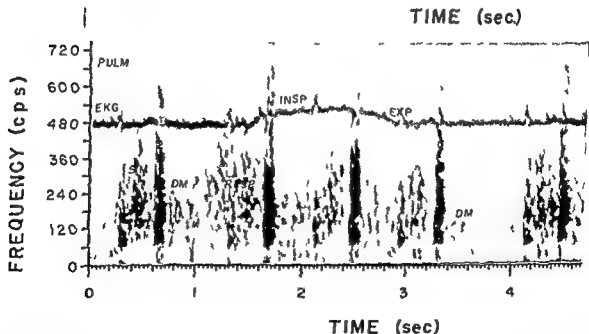
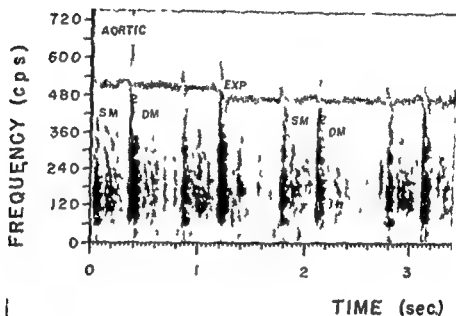
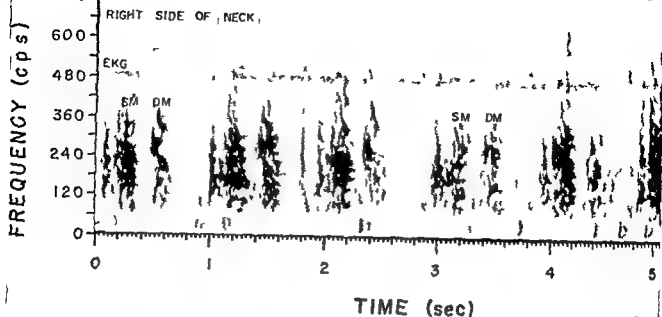


FIG 463 Severe and chronic anemia

Areas indicated in T C (B21255 aut 27582) 13 year old female with chronic severe anemia of the familial Fanconi type (12514). In the neck (top) there are murmurs which may be partly of arterial partly of venous origin. The component related temporally to ventricular systole is probably arterial in origin in the main whereas the diastolic one is venous. In the aortic area (center) there are systolic and diastolic murmur. In the pulmonary area (bottom) S is considerably accentuated and displays normal inspiratory splitting. The systolic murmur has the pattern of an ejection murmur (p 194). There is a decrease of early diastolic murmur. The heart murmurs were normal at autopsy.

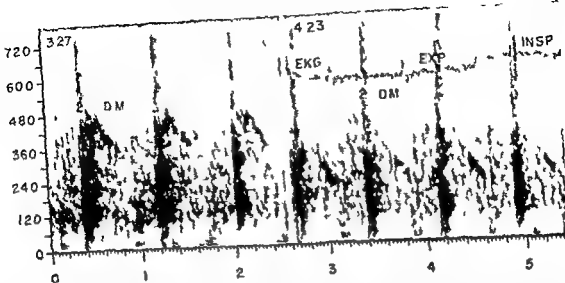


FIG. 466. Mitral diastolic murmur of SBE.

Lungular area in J. B. (732861) 3<sup>rd</sup> years old with SBE of the aortic valve caused by *Streptococcus viridans* on the basis of previous rheumatic affection. The murmurality superimposed on the conventional noise murmur appeared midway in the course of therapy. There is a single harmonic with a crescendo-decrescendo pattern in the murmur of retroverted cup. In the later record the harmonic occurs only in late diastole probably in relation to atrial contraction whereas in the earlier record the harmonic stopped abruptly at about the time of atrial systole & still later recording shows no murmurality.

heart endocarditis is likely to cause murmur probably because of the lower pressures to which the valves are exposed. For example the incidence of murmurless bacterial endocarditis of the pulmonary valve is higher than in the case of the aortic valve. In a series of 23 cases of right-sided bacterial endocarditis a significant murmur was absent in 12 (44%). In all but one the correct diagnosis was not made.

It is a little rule not always remembered however that the occurrence of murmur and unexplained fever of more than a few days duration calls bacterial endocarditis until otherwise proved. In such cases in adults acute rheumatic fever presents one of the most difficult problems in differential diagnosis (1313).

The incidence of involvement of heart valves by SBE displays the same order as to relative frequency as for rheumatic valvulitis: mitral aortic tricuspid pulmonary (634). Acute bacterial endocarditis may affect previously normal valves. In the mitral valve rheumatism the most frequent basis of change leading to SBE. It is mild affection of the valve and of the heart which is most often complicated by SBE. The usual story is that of a patient who either has known of no cardiac affection or has had an asymptomatic

typical aortic murmur for many years. The rarity of the complication of SBE in a patient with atrial fibrillation is another expression of the same fact. In part the relative rarity of SBE in severe rheumatic heart disease is more apparent than real the severe cases are fortunately much less common than the mild cases severely affected patients die early and are not exposed to the hazards of intermittent bacteremia for such a long time. Congenital bicuspid aortic valve is frequently the site of SBE. The tricuspid valve is affected in mainliners morphine addicts who take narcotic by the intravenous route using crude non-sterile techniques. The tricuspid valve may be involved in rare cases of anomaly of the valve. The pulmonary valve is affected relatively frequently by the gonococcus.

The murmur of regurgitation at mitral valve may have a musical element (Fig. 466) when the regurgitation is on the basis of bacterial endocarditis. Penetration of a cusp or tear of a cusp probably results in a member which is free to vibrate musically. Such a vibrant member appears to be a *sine qua non* for the production of musical murmurs in the cardiovascular system. I have a patient (J. B. 170615) with a musical systolic murmur of mitral regurgitation—fifteen years

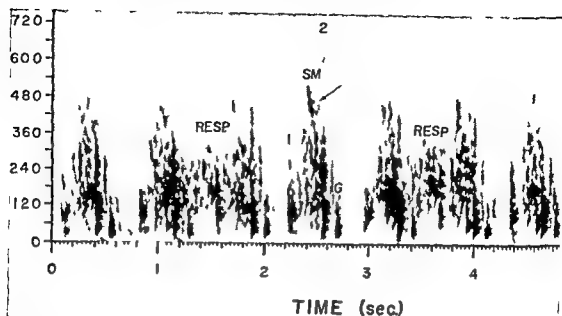


FIG. 465 Mitral regurgitant systolic murmur in C C (681160) 14 year old patient with acute rheumatic fever. Note the third heart gallop and the faint mitral opening snap between  $S_1$  and  $S_2$ .

appeared 2 months and 7 months later. Leimstem and Dimassi (4551) comment on the rather frequent occurrence of an early diastolic murmur at the base in acute rheumatic fever and the subsequent disappearance of same. Usually in their experience, such cases later show evidence of permanent cardiac damage and they emphasize the paramount prognostic significance of auscultatory findings.

In 1951 Besterman (98) suggested the use of a vasopressor agent specifically phenylephrine to aid auscultation of the diastolic murmurs of early rheumatic valvulitis. The elevation of diastolic pressure seemed responsible for the accentuation of aortic diastolic murmurs and probably reflex bradycardia with increased mitral flow accounted for the improved audibility of the mitral diastolic.

The myocardial involvement of ARI is likely to result in dull heart sounds and gallops. Whenever there is myocarditis, conduction defects most often prolongation of the PR interval may occur. Many times it is possible to follow the clinical course of the patient by the intensity of the first sound which is inversely related to the PR duration. Coombs (293) stated: "In very acute carditis the first sound at the apex may become softened and almost inaudible", he ascribed the change to myocardial weakness. Keith (775) was early to point out the more intimate relationship to PR duration.

Pericardial friction rubs and residual systolic clicks are the usual expressions of pericardial involvement. It is a well founded axiom that rheumatic pericarditis without endocardial murmurs is rare. The late systolic click followed by the normal  $S_2$  can suggest  $S_3$  followed by opening snap. Similarity with the late systolic click is a sequel of ARI usually is enough to make the differentiation easy. It can be noted that it is the first of the two sounds which has the snapping dry quality. It is too early in the clinical course for an opening snap to have developed. The phonocardiogram assists in the differentiation. The extra sound is too close to  $S_2$  to be an  $S_3$ . The true  $S_2$  may be identified by the occurrence of normal inspiratory splitting. The SPCG displays differences between valve closure sounds on the one hand and clicks or snaps on the other.

Feinstein (4551) comments on the frequency with which diastolic murmurs are missed in acute rheumatic fever. In the experience of his group when careful and frequent auscultation is practiced no other single sign is of as much prognostic significance as the auscultatory findings.

#### BACTERIAL ENDOCARDITIS

A murmur due to regurgitation at the involved valve is the most frequent finding. Cuts of subacute bacterial endocarditis (SBE) without any murmur of any sort are reported (253, 780) but must be very unusual. On the right side of the

with production of a mural aortic dissection murmur

Logue, Brinn and I (1951) have observed a group of patients in which cystic medial necrosis of the ascending aorta with progressive dilatation and subsequent dissection occurred in association with calcific aortic stenosis of moderate severity. In all cases there was at least mild aortic regurgitation as well. We suggested that the valve lesion produced hemodynamic change which resulted in cystic medial necrosis in the ascending aorta as a non-specific morphologic expression of stress. It is a less likely possibility that cystic disease in the aorta and aortic valves was primary and was the common denominator in the calcific aortic valve disease and aortic aneurysm.

Valvular murmurs due to partial obstruction should be sought over the arteries, particularly at points near their origin from the aorta. Double Korotkoff sound over the right brachial artery but not over the left were noted by Calbraith and Norman (1958). Murmur should also be sought over the back. Evans and Curry (1938) found a grade III aortic murmur along the dorsal spine maximal at about the level of the angle of the scapulae.

Pericardial friction rub may occur from leakage of dissecting aneurysm into the pericardial sac as demonstrated whenever air is introduced into the pericardial sac as in cases of pericardial effusion the pericardial reflection extend very high on the aorta usually to the take-off of the innominate artery. It is probable that a fibrinous reaction with friction rub can be induced on the surface of the aorta when there is dissection in the media without there being actual rupture into the pericardial sac. Whether pericardial effusion of significant proportion can occur on this basis is not certain. In addition it is likely that actual leakage of blood into the pericardial cavity can occur and the patient survive for months or even years thereafter (1942). The clinical picture of both of the above phenomena may resemble a benign idiopathic pericarditis. Pericardial friction rub is an important sign in both types of pericardial involvement.

Syphilitic aortitis is rarely accompanied by dissection but aortic aneurysm of course occurs commonly. It is likely to be timorous

(216). The like many of the other features of syphilitic aortitis such as calcification in the media of the ascending aorta extending to the sinuses of Valsalva and occlusion of arterial branches at the arch of the aorta and of the coronary ostia probably due to secondary intimal atherosclerosis to which the disease of the media renders the aorta exceedingly prone. The "wooden" quality of A is probably due to fibrotic and atherosclerotic change in the aortic cup. Widening of the base of the aorta with closure of the aortic cup through a greater than normal excursion may be a factor it is probable that the base of the aorta can dilate to a certain extent without development of aortic regurgitation because of compensatory stretching of the aortic cup or adequate filling of the orifice because of excessive size of the normal cup.

#### EMBOLISM

Air embolism has been reported rather infrequently mainly because its occurrence usually indicates an accident for which the physician may be responsible. From what clinical information is available and from observations in experimental venous air embolism in animals a loud churning sound or mill wheel murmur is to be expected. It may be audible to the naked ear (191). It is important to recognize the pathognomonic sign since turning the patient into the left lateral decubitus may be life saving (191). Trapping of the air in the right atrium (1163) appears to be the mechanism of the beneficial effects which have been proved in human cases as well as in dogs. Aspiration of the heart has also been practiced (1432).

Hamby and Ferry (626) in an article pointing out the risks of air embolism in neuro-surgical operations performed with the patient in the sitting position described a patient in whom the mill wheel murmur disappeared promptly when he was turned into the recommended position and Muirgrove and MacQuigg (1114) described a similar experience. Duboczky (373) described air embolism with mill wheel murmur audible to the unaided ear occurring during induction of pneumothorax. The sound ceased at once when the patient was turned in the left lateral decubitus position.

after SBI—caused by streptococcus viridans and cured with sulfadiazine. I ven the murmur of mild mitral stenosis has a musical element in this patient.) The musical element is often changeable and may disappear a few weeks or months after it first develops (247, 1112). The abrupt appearance of a musical element suggests fenestration or tearing of a valve cusp. Porter (1219) was listening to a patient in rupture of an aortic cusp occurred. It is, of course, a cusp of the aortic valve which most often ruptures, however, involvement of the inferior (aortic) leaflet of the mitral valve with rupture and generation of a musical apical systolic murmur also occurs—witness the case of I F N. (19722).

Although a musical element may be changeable, the murmurs in bacterial endocarditis are, on the whole, less changeable than is usually taught. "Changing murmur" should not be insisted upon for the diagnosis.

Lillehei found experimentally that bacterial endocarditis is likely to occur when bacteria are injected into the blood stream of animals with systemic arteriovenous fistulas (918). The heart valves are involved (Bacterial infection of arteriovenous fistulas may occur of course in man (633) and in animals.) I have observed one patient (I McC 503929) in whom the Lillehei phenomenon appeared to be present: there was a peripheral arteriovenous fistula and bacterial endocarditis developed on the mitral valve. Curtin, Peterdorf and Bennett (321) described a similar case from the Johns Hopkins Hospital—a 26 year old housewife (I T 411128 aut 20932).

Tuberculous endocarditis has been reported

#### DISSECTING AND OTHER TYPES OF AORTIC ANEURYSMS AND SYMPHYTIC AORTITIS

The varieties of musical murmurs which may occur in dissecting aneurysm have been outlined on page 220. Their abrupt appearance can be a valuable clue to the diagnosis. Roberts (1284) reported a case which demonstrated in addition to the usual aortic diastolic murmur (see below), a systolic thrill of peculiar vibrating nature and a long, coarse, *whistling* systolic murmur in the aortic area and neck vessels. At autopsy he found a prominent shelf-like projection in the aorta just above the valve. *Not a systolic murmur is com-*

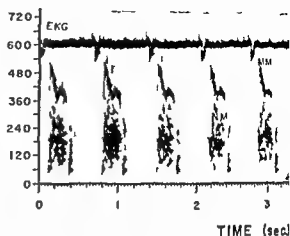
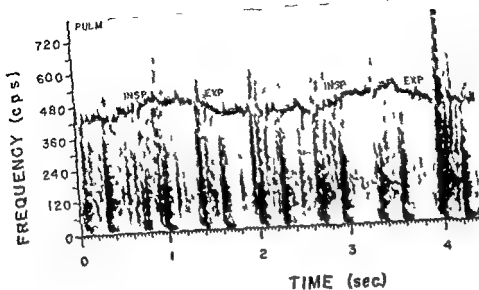


FIG 467 Dissecting aneurysm

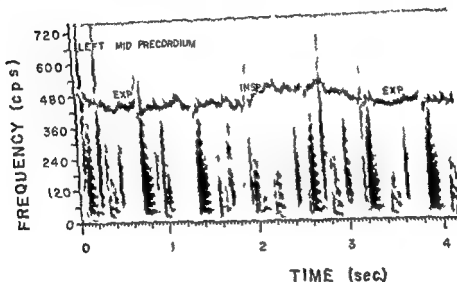
Recorded in localized area at base of neck on right in A H (714091) with dissecting aneurysm and partial occlusion of the innominate artery. The peculiar contour of the harmonic is puzzling.

monly heard over the ascending aorta in case of dissecting aneurysm.

The appearance of an aortic diastolic murmur is another useful diagnostic clue (1424). The sign was first reported in the Anglo-American literature in 1925 by Resnik and Keefe (1262) who attributed the murmur to backflow in the fibrous channel. In 1933 Louis Himmman (628) used this sign to make the clinical diagnosis of dissecting aneurysm—in a CPC to be sure—and suggested the now generally held explanation that distortion of the aortic ring, by the medial hematomata results in aortic regurgitation. Detachment of an aortic cusp (891, 973) or lowering of an aortic cusp as a result of the intimal rent in the aorta above (1424) may at times be the mechanism. In many cases—possibly this is more often the case than the appearance of the murmur after dissection—the diastolic murmur indicates the dissection and is on the basis of idiopathic cystic medial necrosis, the Marfan syndrome or hypertension. Schnitzler and Biver (1363) found that in aortic diastolic murmur had been described in 24 per cent of reported cases. In some cases, e.g., two reported by Levinson *et al* (891) the murmur had a musical quality. This is not surprising since it is well known that the aortic diastolic murmur in patients with the Marfan syndrome may be musical. Furthermore, cystic medial necrosis of the aorta without dissection may lead to partial detachment or fenestration (658, 1051) of an aortic cusp



1



2

FIG. 468 Media tinal emphysema

T. R. (1913), 14 year old boy has had bronchial asthma from the age of 2 years. After an upper respiratory infection with cough the patient suddenly developed tearing chest pain. There was subcutaneous emphysema in the neck. Characteristic crunching sounds were heard. The clicks responsible for the crunch (X) are usually mutually equal and are present in diastole as well as systole.

During World War I it was noted (1413) that in penetrating wounds of the chest in the region of the heart there might be heard a sound of the nature of a click which varied from a faint sound heard by careful auscultation to a voice which may be compared to that heard in the earpiece of a telephone when the lever is moved

up and down. It may be heard sometimes when standing at the foot of the patient's bed. Sometimes the patient is conscious of it. Smith (1413) seems to have invented the expression pericardial knock.

In 1923 La ter (934) described a 21 year old man who developed spontaneous pneumothorax



## PULMONARY EMBOLISM

In pulmonary embolism a systolic murmur may suddenly appear over the pulmonary artery. The mechanism is probably most often acute dilatation of the pulmonary artery and infundibulum. On rare occasion it is possible that the embolus itself, lodged in the pulmonary artery or even the valve area, is responsible. Bunn (191) described a 55 year old woman who suffered multiple pulmonary embolization after the sclerosing of varicose veins of the legs. While he was listening, "there was the onset of a most unusual cardiac murmur. It was a harsh, grinding, continuous sound which varied in intensity but continued through systole and diastole. The intensity of the sound was greatly accentuated with systole. It had somewhat the character of an arteriovenous aneurysm in that it was continuous and rather roaring." The murmur had disappeared a few hours later at which time the patient had shock, hemoptysis and signs of pulmonary consolidation. She died 48 hours after the murmur was heard. Warburg (1908) had a possibly comparable experience. In a man with long standing, rheumatic heart disease and atrial fibrillation whom he had many times examined, Warburg described the following episode: "During the last 24 hours he had heard a sound from his chest which he described as though something were dripping. He said that he thought his heart had burst. His wife was able to hear the sound when she was lying in the bed beside him. I was able to verify his statements. At every heart beat a clicking or slightly sonorous sound was audible in the room. A phonocardiogram showed that besides the sounds usually found in this patient there was a new murmur partly during systole partly immediately after systole. Four days later he had a typical pulmonary infarction with hemoptysis, stitch and fever. As soon as the infarction occurred he was no longer able to hear the sounds and when I examined him the same afternoon the very loud new sounds had disappeared. He recovered in about ten days and since then I have had the opportunity of examining him numerous times but I have not heard the clicking and sonorous sounds. Most probably he had a loose thrombus in the right ventricle which later passed into the pulmonary artery."

McGinn and White (1965) wrote as follows in

describing a series of cases of acute massive pulmonary embolism: "In two cases pericardial friction rubs were loudest in the second left inter space and are interpreted by us as probably due to the dilated pulmonary artery or distended right ventricle rubbing against the pericardium or with the pericardium pressing against the anterior chest wall."

# INFUNDIBULAR DIAHRAGMATIC HERNIA MEDIASINAL EMPHYSEMA, PECTUS EXCAVATUM AND OTHER CHANCES IN STRUCTURES NEIGHBORING ON THE HEART

(including a discussion of some of the cardiovascular sounds which may be audible to the naked ear at a distance from the chest)

Systolic murmurs occur commonly with *pectus excavatum*. As to mechanism, distortion of the right ventricular outflow tract, pressure on a lappet of lung, and rubbing of pericardial surfaces normal or abnormal are all possible. Circumscribed systolic timing or extension of the murmur across the second heart sound are features suggesting a pericardial or cardiopulmonary mechanism.

Diastolic murmurs with *pectus excavatum* must be viewed with more suspicion. Conceivably distortion of the pulmonary valve area might be responsible. Since *pectus excavatum* of severe proportions can occur as one feature of the Marfan syndrome (1971) other stigmas of this disorder should be sought particularly when a diastolic murmur is present. I have information on two patients (840) without definite evidence of the Marfan syndrome in whom a diastolic murmur of arterial type persisted after surgical repair of the sternal deformity.

In his delightful essay on precordial noises heard at a distance from the chest' (74-75) William Bennett Bean reviews many of the sounds produced by the action of the heart on surrounding structures which are in one way or another altered. These conditions include pneumothorax (traumatic therapeutic spontaneous) interstitial emphysema of the lung and mediastinum (spontaneous or traumatic), ectopic barboarygmia (as with diaphragmatic hernia or with aneurysm of the aorta) hydropericardium (see p 416)

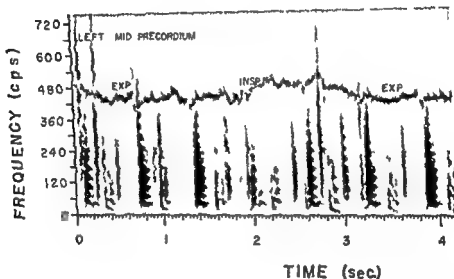
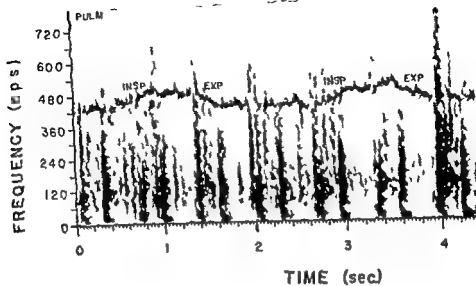


FIG. 15. Metallic empty ems

T. R. (19018) 31 year old boy has had bronchial asthma from the age of 2 years. After an upper respiratory infection with cough the patient suddenly developed tearing chest pain. There was subcutaneous emphysema in the neck. Characteristic crunching sounds were heard. The clicks responsible for the crunch (X) are usually musical and are present in diastole as well as systole.

During World War I it was noted (1413) that in penetrating wounds of the chest in the region of the heart there might be heard a sound of the nature of a click which varies from a faint sound heard by careful cultivation to a noise which may be compared to that heard in the carpiece of a telephone when the lever is moved

up and down. It may be heard sometimes when standing at the foot of the patient's bed. Some times the patient is conscious of it. Smith (1413) seems to have invented the expression pericardial knock.

In 1928 Lister (934) described a 31 year old man who developed spontaneous pneumothorax

"As he sat giving the history of his illness, at a distance of some five feet a hollow knocking sound could easily be heard coming from the patient's chest." It was loudest at LLSB in expiration, and in the upright position, resembled the Korotkoff sounds in quality, and was mid systolic in timing. Frost and Bing (499) described a case in which the extra sound "most resembled the clicking of the tongue used to urge on a horse." Scadding and Wood (1353), Sharpey Schaffer (1380), Edwards and Simpson (414), and others have reported on similar cases. Occurrence of the pneumothorax on the left seems to be a constant feature.

Mediastinal emphysema, although occasionally remarked upon by earlier writers including Irenæus (see p. 8), was brought to clinicians' attention mainly by Louis Hamm in the late 1930's. The severity of the chest pain, suggesting myocardial infarction makes the condition of im-

portance to cardiologists. Curious sound, synchronous with the heartbeat and occasionally very loud, are frequent in such cases (Fig. 468). The sound has been compared to the "wadding up of paper" (1381), to "a crunching crackling sound like small chicken bones being crushed" (1209), "the noise that one hears on crumpling a handful of cellophane close to the ear" (952). The sounds are frequently audible at a distance of several feet from the patient. The phonocardiogram (693) in mediastinal emphysema shows a succession of clicks in both systole and diastole (Fig. 468). There is essentially complete lack of pattern to the timing and intensity of the clicks, unlike those of pleuropulmonary origin.

On the basis of personal experience in 7 patients and a review of 98 cases of so diagnosed spontaneous mediastinal emphysema reported in the literature, Scott (1370) raises the question of

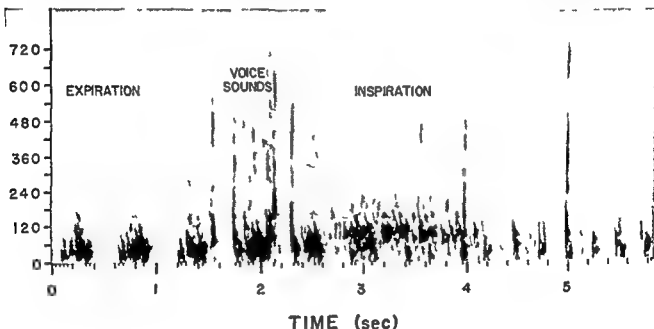


FIG. 469 Systolic murmur caused by pulmonary artery compression

Here is shown a continuous recording from the pulmonary artery of a patient with lymphosarcoma of the mediastinum on the left in the normal expiratory chest position and on the right in inspiration. A harsh systolic murmur present in expiration disappears with inspiration. The artifacts in the recording are spoken voice sound and breath sounds. Note the disappearance of the murmur with inspiration. Note further the splitting of the second sound which appears with inspiration. The murmur in this patient is believed to have been the result of pressure of a lymphosarcomatous mass on the pulmonary artery. Inspiration by increasing the capacity of the chest removed this compression. The murmur in this case has the typical appearance of an ejection stenosis murmur which to be sure it is. Its peak occurs later in systole than the peak of the ejection stenosis murmur of aortic origin for instance. In some reported cases cardiovascular disease has been simulated by mediastinal tumor. Pulmonary artery compression has been proved to be the cause of the systolic murmur and the murmur has disappeared after removal of the tumor (499A, 1574A).

whether many of these cases may not in fact be instances of shallow left pneumothorax without mediastinal emphysema. He argues convincingly that the rucultatory phenomenon in the two conditions may be identical. Subcutaneous emphysema and a ray evidence of air in the mediastinum were lacking in many of the cases. On the other hand in 45 of the 98 reported cases a small left pneumothorax was described. In only four was there a right pneumothorax. Pertinent to this discussion is the mechanism of pneumothorax whether it arises first as interstitial emphysema, as believed by the Micklin (1914) or through rupture of a subpleural bleb. Chapman (219) has also raised a question of the specificity of Hamman's sign for mediastinal emphysema.

Possibly the circumstances in Hamman's first case (631) are consistent with origin of the adventitious sounds in a shallow left pneumothorax alone rather than in mediastinal emphysema.

The patient laughingly said he could reproduce the sound. He turned on his left side, huffed about a few moments and suddenly said: "There it is." I put my stethoscope over the apex beat of the heart and with each impulse heard the most amazing sound. It is difficult to describe. Crunching is the best adjective I can think of though it is far from apt especially since crunching has been widely used to describe plural frictions to which it bears no resemblance. I certainly conveyed the impression of air being churned or squashed about in the tissues. When the patient turned on his back the sound at once disappeared.

Allen (17) described an unusual case of huge diaphragmatic hernia in which the patient, a woman, came for examination complaining of heart trouble because of a disturbing, noisy, synchronous with the heart beat and present intermittently for two years. It was especially bothersome in bed at night and was specifically described by the patient as resembling the plashing of a water wheel. Green (191) found that introduction of air into the colon in amounts sufficient to distend the pleural sac would result in a metallic knocking noise. Sometimes a stolic sometimes a diastolic. Roberts (1283) and Bean (74-75) have pointed out that splashing or knocking sounds may be audible when the stomach is filled by a proper mixture of gas and fluid and the heart is active. The circumstances may obtain in normal individual after a large meal

especially if the subject is seated slouched in an upholstered chair or in a neurotic individual with excessive hyperactivity and air swallowing it may be the basis of puzzling symptomatology. The normal individual may be able to abolish the sound by belching.

Bean (74-75) describes the observation of one of his associates, Dr. Lewis I. January, who studied a healthy young man with the peculiar ability to make his heart sounds audible to a distance of several inches when he held his mouth open. There was no evidence of heart disease or of abnormality of the lungs, esophagus and upper GI tract. It was suggested that through some anatomical or functional quirk the subject kept the esophagus patulous when the mouth was open and that the esophagus behaved like a megaphone.

Diaphragmatic flutter (373) is a condition with several features suggesting heart disease. Pain in the chest, shoulder and abdomen simulates angina pectoris. It has been called diaphragmatic angina. The patient may complain of palpitation. On auscultation a to-and-fro-hustle like a pericardial friction rub or a tick-tock-tapping, swinging or churning sound may be heard. It should be possible to distinguish the sounds from any of cardiovascular origin by the fact that the rhythm is distinct from that of the heart. The adventitious sounds are heard well away from the precordium and over the lower part of the chest. The phenomenon persists in sleep. Diaphragmatic flutter occurs in pyrexia, is often cryptogenic and is rare—much rarer than might be thought from the number of reports in the literature. One famous patient has been reported at least 15 times beginning with William Branch Porter (1226) in 1916 (214-15) and 1943. Intercostal flutter is even rarer. Diaphragmatic and intercostal flutter are varieties of respiratory tic.

### IRREGULARITY

The murmurs of pre-existing heart disease (congenital or rheumatic for example) tend to be altered so markedly during pregnancy that evaluation of their functional significance may be difficult and risky. On the other hand pregnancy affords an excellent opportunity for the detection of heart disease (2023). Pregnancy is the only

occasion for many young women to have a physical examination. 2) By accentuating diastolic and systolic murmurs pregnancy will facilitate the diagnosis of heart disease, although precise evaluation of its significance may have to wait delivery.

A systolic murmur in the pulmonary area occurs commonly in pregnant women probably on the basis of increased stroke volume and possibly displacement of the heart. Systolic clicks may develop on the latter basis just as they may be heard in massive ascites—displacement of the heart may cause it to press on the anterior rib cage with movement of costochondral and/or chondrosternal joints.

Burwell and Metcalfe (1924) have, contrary to the stated experience of others (1911), never heard a basilar diastolic murmur in pregnancy in a patient who could be considered free of heart disease. A murmur of the Carey-Coombs type at

the apex probably does not occur in physiological pregnancy. For it to occur unusual anemia, rheumatic carditis, or a congenital malformation must be present.

In late pregnancy and early after parturition a mammary souffle (see p. 233) may be heard over both breasts, usually at the upper border. The murmur may be only systolic, specifically late systolic, or may be continuous. The facts that it is obliterated by pressure on the stethoscope and that it is confined to late systole, when combined with an acquaintance with the phenomenon, will usually suffice for identifying the murmur.

Venous hums occur commonly. Pitting edema over peripheral arteries are not uncommon.

The uterine souffle, fetal heart sound and other sonic phenomena related to the uterus and its contents have been described on pp. 233 and 255.

## SECTION V

*Physiologic, Pharmacologic, Physical and Other  
Procedures Employed in the Investigation of  
Cardiovascular Sound in Man*

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## CHAPTER 22

# Procedures for Study of Cardiovascular Sound in Man

### With a Section on Quantification

As noted on p. 101 phonocardiography and the study of cardiovascular sound in general require great significance when quantitation is possible and when they are elevated from the realm of pure observation to that of an experimental science by the designed use of various test procedures. It is the purpose of this section to review and analyze what has been done in this area.

**RESPIRATION** (173, 831) The normal inspiratory splitting of  $S_2$  (p. 150) the paradoxical splitting (i.e. splitting in expiration) which occurs under certain circumstances, the exaggeration of the murmur of tricuspid regurgitation and re-augmentation during inspiration (p. 324) are useful in the study of patient.

Maronde *et al.* (1943) have pointed out the existence of a paradoxical paradoxical pulse in left ventricular failure. The pulse murmur in inspiration rather than decreased in inspiration. The phenomenon appeared with compensation. Parallel change in the intensity of murmur of aortic and mitral valve disease might be expected although the magnitude of the change is probably light.

**THE VASCULAR MANEUVER** During straining the increase in intrathoracic pressure causes an immediate reduction in venous return to the heart. The output of the right heart decreases as the pulmonary reservoir is depleted and the output of the left heart falls. With relaxation the venous blood which was dammed back peripherally rushes into the heart. The pulsations in the pulmonary artery, previously reduced in amplitude become vigorous at once according to information supplied

by electrokymography (772). The output of the left heart and aortic pulsation become maximal only after refilling of the pulmonary reservoir.

Zimmer and Kay (1946) found that the behavior of murmurs after the release of Valsalva type straining is useful in distinguishing right-sided and left-sided lesions. Most murmurs were diminished sharply by straining. In the case of right-sided lesions the murmur returned promptly with cessation of straining whereas a delay was observed with left-sided lesions. The murmur of pulmonary AV fistula returned early. Functional pulmonary systolic murmurs behaved like right-sided lesions.

Mitral stenosis and patent ductus arteriosus behaved in a modified but characteristic manner. The mitral opening snap often disappeared with straining and returned a few cycles after release of straining. The early diastolic portion of the diastolic murmur behaved in a manner typical of left-sided lesions. The presystolic murmur varied more as a function of heart rate than of the changes in volume of flow which were presumed to accompany straining. Because of the tachycardia during straining the presystolic murmur might be increased paradoxically in that period and be decreased with the bradycardia of the post-strain period.

The murmur of patent ductus arteriosus was moderately reduced by straining. Interestingly in the early post-strain period further reduction in the murmur usually occurred. The increased pulmonary flow at that time apparently reduced flow in the shunt.

As one might expect,  $P_2$  became split in the



early post strain period Zimser and Lutz (1606) thought the first element of the split was pulmonary closure sound—a view difficult to reconcile with other information—mainly because this element seemed to be accentuated early and the second element accentuated late in the post strain period.

In experimental murmur producing lesions in dogs, Bertrand and collaborators (96) confirmed the clinical observations. However, observations on the Valsalva maneuver in heart failure (377, 807, 1379) leave some doubt as to the validity of the test when central blood volume is increased. Specifically, in heart failure no change in pulse pressure and no post straining "over-shoot" was observed. No change in murmurs can therefore, be anticipated. Practically speaking, patients in heart failure are often too dyspneic or too sick and weak (1603) to perform the test adequately. Goldberg, Flisberg and Katz (363) found that in mitral stenosis of only acoustic significance the response to the Valsalva maneuver was identical to that in normal in "over-shoot" of blood pressure and bradycardia in the post strain period. In severe mitral stenosis these normal phenomena were abolished (390). With straining there is a drumming up of blood in the systemic venous system. With release of straining venous return to the heart increases markedly through release of this dam assisted by the hyperpnea of the post strain period. The increased cardiac output accounts for the "over-shoot" in blood pressure which in turn produces the bradycardia by reflex mechanisms. In severe mitral stenosis mitral flow cannot increase sufficiently to produce these changes. In those few healthy individuals who for some reason can maintain blood pressure during the Valsalva autonomic block with tetraethyl ammonium chloride (590) will return the pattern to "normal" whereas the patients with significant mitral stenosis maintain brachial pressure during the Valsalva despite IFAC.

With the Valsalva maneuver Lee and Gimlette (867) produced partial reversion of the shunt in ASD. The effect on splitting of the second sound should be investigated. Reversal of the left to right shunt occurs during the post straining period. During straining the left to right shunt may be increased.

**TOURNIQUETS** Leonard and colleagues (876) have demonstrated the usefulness of the application of tourniquets in the study of gallop. The effect of impounding of blood in the extremities (819) may have usefulness in connection with other sonic phenomena.

**EPINEPHRINE, NORTHAEPHRINE, AND RELATED SUBSTANCES** Epinephrine has been shown to abbreviate the isometric contraction period of the ventricle (801, 802). The pre-isometric period is probably shortened also. The result is that  $S_2$  is accentuated by the more rapid closure of the AV valves and becomes sharper through the addition of components of higher frequency. The  $QS_1$  interval is reduced. The phenomena may occur with epinephrine in dosages insufficient to affect blood pressure. Stimulation of the sympathetic nerves to the heart (or section of the vagi) has a similar effect (1551).

With larger doses of epinephrine the second sound likewise becomes accentuated. The effects produced and the changes observed in the heart sounds are related to both the greater and the lesser circulations.

With epinephrine the Korotkoff sounds are intensified (271). Lewis and Hewlett (889) concluded by comparing the effects of nitroglycerine that the effect was the result mainly of increase in the stroke output.

Because of increased velocity of blood flow, any systolic murmur—including the functional pulmonary systolic murmur of young subjects—is likely to be exaggerated by adrenaline. Holldack and Mickle (703) heard and recorded a diastolic murmur in the third and fourth left inter-space after adrenaline. It had the timing of a mitral diastole but the quality of an aortic. They thought it was produced by the pulmonary valve.

Posterior pituitary extract was shown by Wiggers (1350, 1352) to increase systemic arterial pressure at the same time producing a fall in pulmonary arterial pressure. He further observed in dog (1348) the expected changes in A and P. Posterior pituitary extract has little or no effect on blood pressure in man (3883) however.

Amyl nitrite has been suggested for eliciting the murmur of mitral stenosis (888) for identifying functional aortic insufficiency (126), and for differentiating between aortic stenosis and mitral

regurgitation (38). Crum (326) claimed that 14 of 200 autopsied cases of hypertension showed minimal aortic regurgitation of relative type and nitrite abolished the murmur by reducing diastolic pressure. Barlow and Shillingsford (38) suggest that nitrite for differentiating regurgitant from ejection stenotic murmurs. Atrial nitrite reduces diastolic pressure and effect is decreased in a mitral regurgitant murmur it increases cardiac output and increases in ejection stenotic murmur (e.g. that of aortic stenosis).

The breathing of mixtures containing reduced amounts of oxygen is accompanied by increases in pulmonary artery pressure (112). The effect on murmur with that of patent ductus arteriosus or pulmonary regurgitation may be diagnostically helpful. Monheim (1032) administered 9 per cent oxygen mixture to four patients with patent ductus but it is not clear from his report what happened to the murmur.

**Position.** The head-down position can be used to accentuate atrial heart sounds (43). The recumbent posture is the best for detecting the physiologic third heart sound especially in the prone decubitus the first assumption of the recumbent posture. The left lateral decubitus and sitting leaned forward postures are usually optimum for visibility of the murmur of mitral stenosis and aortic regurgitation respectively. The knee chest position is sometimes useful in hearing the murmur of aortic regurgitation. To detect a pericardial friction rub it may be necessary to place the patient in many different positions.

In 1902 Gordon (373) found that the venous hum disappeared in the recumbent position but hearse murmur and the murmur of tricuspid regurgitation mitral regurgitation and aortic stenosis increased. The murmur of aortic regurgitation showed little change.

**CARDIAC CATHETERIZATION OR (1377)** Aside from important contribution toward establishing the diagnosis in many instances cardiac catheterization provide or may eventually provide four main types of information useful to the study of cardiovascular sound (1) volume of blood flow including difference in stroke volume of the two ventricles in the case of heart (2) intracardiac pressure (gradients across heart valves and septal defect) the time course of pressure changes

for correlation with the type of murmur (nature of mechanical events) for identification of nature of transient e.g. in opening sound of the arterial valve (3) the velocity of blood flow (400-401-1132) (4) in combination with appropriate injection of a dye or other indicator and sampling from other sites catheterization may eventually permit quantitation of valvular regurgitation. In addition of course intracardiac phonocardiography with a catheter microphone can be combined with conventional catheterization method.

The pulmonary capillary (wedged) pressure is useful in differentiating functional from organic mitral stenosis. In a patient with aortic regurgitation due to rheumatism Wade, Litch and Werko (1495) were able to identify the regurgitant apical diastolic murmur as an Austin Flint by the fact that pulmonary capillary pressure was normal both at rest and with exercise. By the findings of an elevated PC pressure in a patient with exerciseable cellanemia (1070) was able to establish that the apical diastolic murmur resulted from organic mitral stenosis a fact confirmed by autopsy.

Ever since the method was first introduced a century ago by Chauveau in collaboration with Marey and others (see p. 12) the timing of event in both sides of the heart by simultaneous recordings of intracardiac and aortic pressure just outside of the heart outlet has been important in elucidating the origin of the sound. Right heart catheterization in man—and more recently left heart catheterization—have contributed precise information on the time sequence (116-170-276-570-573-572).

The recording of heart sound during catheterization ensures that the correlation of cardiovascular sound with hemodynamic are reliable. Changing hemodynamics by maneuvers such as exercise breathing of reduced oxygen mixtures pharmacologic agent is now routinely practiced in combination with cardiac catheterization. Correlation with the changes in heart sound and murmurs should be investigated more extensively.

Pre-recording of cardiac catheterization of both sides of the heart or at operation by puncture of the proper vessel or chamber can be useful in determining the pressure gradient across

stenotic orifices, regurgitant orifices, septal defects, etc. Gordon and his co-workers (570, 571) have demonstrated the usefulness of simultaneously recorded pressures using equisensitive transducers and the same base line in the recordings. It is entirely feasible to have a continuous recording of pressure differential, that is, the difference in pressure sensed by the two transducers, or, of course, differential pressure can be calculated. Finally, when sound recordings are made simultaneously, the pressure recordings permit identification of various elements of the heart sounds and investigation of the relation between pressure gradient and murmur.

**PHYSICAL EXERCISE.** Exercise is used more in connection with eliciting a mitral diastolic murmur of mitral stenosis than in any other single connection. Frequently mitral stenosis of minor grade is missed when a patient is examined while lying quietly in hospital. Even "sit up" may be inadequate for demonstrating it. In the same patient, ambulatory under the conditions of an outpatient clinic, the murmur of mitral stenosis may be very evident. A physiologic third heart sound is likely to be brought out by exercise of the legs in the recumbent posture. Linn and Hubert (927) found that exercise would bring out a presystolic gallop in persons such as hypertensives who did not previously demonstrate it.

Bortor and Muller (138) found that with exertion the QS interval was shortened more than the Q1 interval of the electrocardiogram. The Q1 interval was abbreviated and S1 was increased in intensity.

**RADIOLOGIC METHODS FOR STUDYING TURBULENCE AND (INDIRECTLY) MURMURS.** This is in approach, in which Dotter (364) is pioneering. Occasionally—in the early phases of filling—turbulence may be indicated in angiocardigrams in patients with certain lesions. Usually, however there is a blurring of detail comparable in the vivid analogy drawn by Dotter (365) to the loss of the detail of movement of individual cars down a city street in a night photograph made with a long exposure.

Necessary for the radiologic demonstration and analysis of turbulence are (1) very brief x-ray exposures, and (2) a flocculent radio opaque medium. Both requirements present practical difficulties

which have not yet been overcome. Dotter (364) states: "When it becomes roentgenologically possible to visualize turbulence, the stethoscope may indeed be administered a 'mortal blow'." Although it is difficult to imagine any diagnostic method involving an injection which would combine the informative and safe feature of the stethoscope, this excessively enthusiastic statement at least indicates a technique which can contribute to our understanding of the generation of sound in the cardiovascular system.

**THE QUANTIFICATION OF REGURGITATION.** Rapid advances are being made in the development of methods for quantitative estimation of the volume of aortic and mitral regurgitation (167). In the future correlations of the volume of regurgitation (along with other parameters such as pressure gradient and geometry of the valve lesion) with the characteristics of the murmur should provide information of theoretical interest and practical value.

### QUANTIFICATION IN CARDIOVASCULAR SOUND

The three parameters of cardiovascular sound have been described in considerable detail but mainly in qualitative terms. The temporal dimension is described with reference to the phase of the cardiac cycle with regard to the approximate relationship of one element to another, and with respect to duration of each element. Frequency is indicated by rough adjectives qualifying pitch. Intensity is graded by approximate systems adequate for most clinical purposes such as the system of Leeman and Levine (481). The largest portion of this monograph has been occupied with a review of the qualitative description of cardiovascular sound in health and disease. Phonocardiography attains its highest precision and greatest physiologic usefulness when quantitation is applied in each of the three parameters of heart sounds. It is the purpose of this section to review what has been accomplished in this domain.

The dimension of frequency can be described briefly since the spectral phonocardiogram accomplishes quantification. In fact such a wealth of information is provided by the spectral phonocardiogram that a problem is created in the handling of the mass of data in such a way that one

record can be compared with another. How, for example, is the average frequency content of the first heart sound in a series of normal subjects to be determined from the spectral phonocardiogram? Furthermore, relating frequency to anatomy and hemodynamics encounters complexities which are not easily unraveled.

Measurements of interval between two sonic elements or between a sonic element and another cardiovascular event and the relating of these elements to independently measured cardiovascular parameters have represented a most admirable demonstration of the use of phonocardiography as a physiologic tool. The following is a list of certain of the quantitative studies of the temporal dimension of cardiovascular sound.

1 The relation of the Q1 (onset of Q wave of EKG) to onset of rapid vibrations of S<sub>1</sub> interval to left atrial pressure use of the Q1 interval as an index of the grade of mitral stenosis (778)

2 The relation of the interval between S<sub>1</sub> and the opening snap to the left atrial pressure use of the S-S interval as an index of the grade of mitral stenosis (64, 66)

3 The relation of the interval between the aortic and pulmonic components of S<sub>2</sub> (AP interval) to the level of right ventricular pressure and therefore the severity of pulmonary stenosis (874)

4 Relation of the QK interval (between the Q wave of the EKG and the onset of the Korotkoff sound) to the preceding RR interval use of this relationship as evidence that stroke output influences pulse wave velocity (1296)

The intensity parameter of cardiovascular sound is possibly the most difficult to quantify. The inaccessibility of the generator and the variability and complexity of the surrounding transmitting tissues are part of the problem. Since cardiovascular sound of equal net intensity may vary widely in frequency composition, measurements which do not take this fact into account are of somewhat limited usefulness.

THE MEASUREMENT OF INTENSITY IN CARDIOVASCULAR SOUND. The problem and the several approaches which have been employed are discussed elsewhere. One method used in the measurement of noises of other types is that described by Churcher and Kang (264, 265). The noise to be quantitated is compared with a reference tone at 1000 cps. The intensity of the reference tone is changed until the two match. The procedure can be done by putting the test noise in one ear and the reference tone in the other or by putting both in both earphones but playing them alternately. The method is comparable to the use of a comparator in colorimetric chemical determination. Contrary to what one might think, it is possible to match wide band noises to pure tones with satisfactory reproducibility. The true intensities such as the first and second heart sound are probably measured only with difficulty and wide approximation by this method but murmurs can be quantitated.

Another and more satisfactory way to measure intensity is to listen with an electronic stethoscope and adjust a calibrated attenuator until the intensity of the sound to be measured disappears (879, 1108, 1319, 1322).



## SECTION VI

### *Cardiovascular Sound in Animals*



## CHAPTER 23

# Cardiovascular Sound in Animals<sup>1</sup>

### TECHNIQUE

To obtain proper electrocardiograms for correlation purposes plate electrodes of the conventional type (with electrode paste) must sometimes be replaced by venopuncture needles passed through the skin and connected to the appropriate lead wires of the EKG machine. Application of the microphone to the chest may be better accomplished by wetting down the hair or even by having it. A larger chest bell than that ordinarily employed may be desirable.

### DISCUSSION

**HORSE** The important role of the horse in the elucidation of cardiovascular sound has been commented on (p. 42) in the discussion of the work of Chauveau. Electrocardiographic studies were performed by Luthoven (417), Kahn (263), Vorr (1146), Neumann Klein and Stefan (1141) and others. The heart rate of the horse is usually between 32 and 40. The P wave of the EKG is interestingly complex, it is usually double (994) and may be even more complex with an additional intrating wave attributed by Vorr (1146) to the sino-atrial node and two late waves which may represent T waves of the two components of the I wave. The PP interval is usually of the order of 0.35 sec.

Luisada and colleagues (994) found no atrial sound except in old or sick horses. When present it began 0.30-0.40 second after the beginning of P. The interval between the heart sound was usually 0.2 to 0.62 sec. In some records of large horses vibration of low frequency were seen beginning 0.10 sec after S occurring therefore during and possibly related to rapid ventricular

sisting. These vibrations listed 0.1-0.2 sec rarely was a third heart sound recorded.

Murk and his colleagues seem to occur rather frequently in the horse (349, 975). Atrial regurgitation was apparently the cause in Duttwiler's (349) 11 year-old gelding which also had atrial fibrillation. Clinical signs of atrial regurgitation and clinical and autopsy manifestations of congestive heart failure were described. A prolonged third heart sound was present.

In two donkeys, Luisada, Weiz and Huntman (994) found a third heart sound.

A typical continuous murmur of physiologic patency of the ductus arteriosus has been described in the newborn foal (184).

**CATTLE** The heart rate varies from 30 to 60 for the bull, 60 to 80 for the cow and 110 to 130 for the calf (180, 1146).

In the record of one bull the second sound was periodically split apparently following the respiratory phase (994).

As in the foal the continuous murmur of PDA has been recorded in the newborn calf (184).

**DOG** The heart rate is highly variable apparently depending on age and sex. Vibrations due to atrial systole have been identified (994).

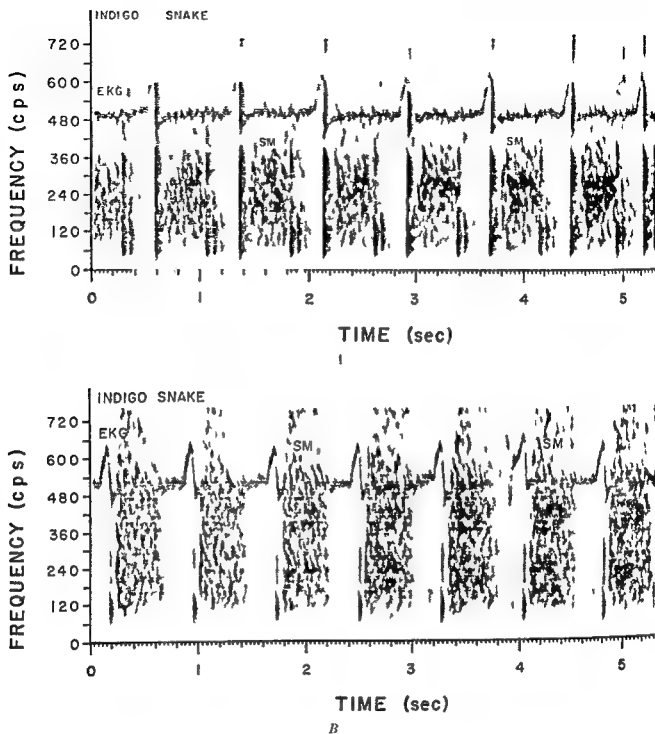
**BUFFALO** Atrial sound were recorded by Luisada, Weiz and Huntman (994).

**DOG** The dog has, of course, been studied extensively in connection with cardiovascular investigations of many types (1399). See Figures 129 and 141 for examples.

**RABBIT AND GUINEA PIG** The heart rate is of the order of 160 for the rabbit and 100 for the guinea pig. Respiratory rate in the rabbit is often about half the heart rate so that in piratory and expiratory sound are easily confused with the heart sounds or with murmurs. In the rabbit as

<sup>1</sup> The assistance of Mr. Arthur P. Wat on, director of the Baltimore Zoo is gratefully acknowledged.





B  
FIG 470 Indigo snake

Recorded from 35th ventral segment in area of apex beat and maximum QRS of the electrocardiogram in 7 foot snake

in the rat and man the second sound occurs at the end of the T wave (585)

RAT AND MOUSE The heart rate in the rat is 400 to 500 and in the mouse 500 to 700 per minute OTHER ANIMALS In the non hibernating hedge hog Johansson (748) and in the kangaroo Sporti

(1426) found a situation opposite to the Heggin syndrome (p 119) In the e animals S occurs appreciably later than the I wave indicating that mechanical systole is longer than electrical systole The elephant's physiology has been studied from several points of view (84) including a description

of the low heart rate of about 30 beats per minute. Apparently phonocardiogram have not been made.

We have recorded the heart sound in frogs, turtle, alligators and snakes. Figure 470 illustrates the finding in 6 to 7 foot snakes (indigo snake *Drymarchon corais couperi* and rock python *Python sebae*) and in 9 foot box constrictors (*Constrictor constrictor imperator*). The QRS of the electrocardiogram, electrical systole and mechanical systole as indicated by the interval between the first and second sound are prolonged consistent with the expected findings in a cold blooded animal. The snake has an interventricular septal defect and has three arterial trunks arising separately from the incompletely divided ventricle: pulmonary artery and two aortae (81A) (476A)

(476B). Each arterial trunk has a bicuspid valve at its mouth (1248A).

The three elements in the part of the cardiac cycle expected for S<sub>1</sub> (Fig. 470A) may be produced by closure of the three arterial valves. The systolic murmur is not as we first thought generated at the ventricular septal defect or elsewhere in the heart and great vessel. It is generated artificially at the skin surface by rubbing of scales between the apex beat and the microphone.

#### GENERAL COMMENT

The duration of the heart sound diminishes and the frequency span of the heart sound increases with diminishing size of the animal and increasing heart rate.



## SECTION VII

*Respiratory Sound (Lung Sound)*



## CHAPTER 21

# Lung Sounds

### PERCUSSION NOTES

The characteristic of tympanitic resonant and dull percussion notes are compared in Figure 411. The three have in common a central core which is the sound of impact of the percussing finger on the plethrometer finger. This impact sound constitutes a considerable part of what one hears in the percussion note. In the case of the completely flat note there is little else because the blow is not able to set the structures under the percussing finger into vibration to any significant extent. In the case of the resonant note vibration, maximal in the range of the natural frequency of the thorax, are superimposed on and follow the impact sound. The natural frequency of the subject thorax (a normal young male adult) is seen to be approximately 140 cycles per second (c.p.).

Comparing the resonant and the dull notes it appears that the former is louder and longer than the dull note is a result of the additional forced vibration of the normal air-filled thorax. The dull note is, on the average (and in the way it impresses the ear) of higher pitch. This is not principally because vibration of higher frequency has been excited but because the average pitch of the dull note is not weighted by the low frequency vibration of the thorax. It is true that in the relatively dull note of pulmonary consolidation vibration of higher frequency and lesser intensity are excited in the pathological tissue which has a higher natural frequency.

Comparison of the tympanitic note (e.g. over the stomach bubble) with the other notes reveals that the main difference is in more musical quality which is reflected in these displays by the presence of more clearly defined harmonics. There is a distinct fundamental at 180 c.p. and the second harmonic at 360 c.p. is equally well defined.

**EARLY STUDIES OF PERCUSSION NOTES** The lesser intensity of the dull percussion note compared with the resonant was demonstrated by Selling (1870) in Friedrich Müller's laboratory by a simple but ingenious method. He compared the maximum distance at which each type of note could be heard when produced by a physician percussing over the lung and over the thigh with equal force. He found that the resonant note is audible approximately five times as far as the dull note. Since intensity is inversely proportional to the square of the distance it can be concluded that the healthy lung excited in the thoracic cage is approximately twenty-five times more resonant than the muscle mass of the thigh. Selling and Scripture also recorded percussion notes on phonograph cylinders of the Edison type and demonstrated by microcopy that the cut produced by the resonant note was considerably deeper than that produced by the dull note. In an even more definitive experiment Selling and Idelm measured the intensity of these notes by means of the Luthoyen string galvanometer and showed that the dull percussion note was of considerably less intensity. The greater duration of the resonant note was likewise demonstrated by Selling's studies by both the phonographic and the Luthoyen galvanometric method.

The timbre of the resonant or vesicular percussion note has been compared with the timbre of the note produced by striking a loaf of bread covered by its crust—the air cells of the bread are analogous to the alveoli. Pitt (1272) observed that when milk is stirred as it is brought to a boil the stirring produces a resonant note when the boiling point is reached. Again the boiling milk contains innumerable tiny bubbles filled with vapor under pressure and separated by

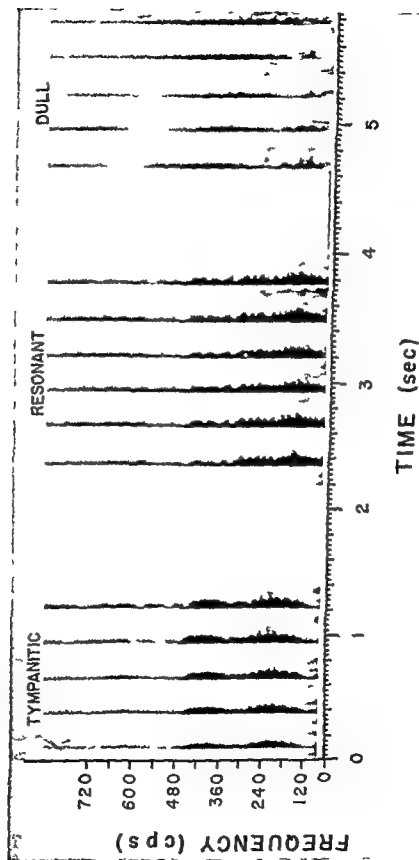


Fig. 471 Percussion notes

liquid partitions endowed with a certain amount of elasticity. Geigel (333) compared the tympanitic percussion note with that produced by percuting a glass cylinder filled with very frothy beer.

Placing the pitch of percussion notes on the musical scale is difficult because they are more nearly noises than musical notes. Nevertheless, an average pitch can be determined particularly by comparison with the pitch of other notes. Just as each of the kettledrums of an orchestra emits a noise of distinct average pitch and a melody can be played on a series of glass tumblers. Auenbrugger and Skoda noted the variation in pitch of different resonant percussion notes, and Austin Flint (466) emphasized this fundamental acoustic characteristic. Gerhardt in 1876 (341) and Selling in 1907 (137a) used Helmholtz resonators for studying percussion sound. Selling found it necessary to use cylindrical conical resonators as much as 20 meters in length to study the lowest pitched of the percussion notes. These studies and those of Martini (1044) and of Ritt (1275) demonstrated a series of tones ranging from a pitch corresponding to F below low C (approximately 80 cps) in the musical scale up to C above high C (approximately 800 cps) and depending on the volume of the thorax (see below). Martini (1044) placed the fundamental of the natural frequency of the child's lung at 170 cps, of consolidated lung at 140 to 190 cps, of normal adult lung at 100 to 120 cps, and of emphysematous lung at 70 to 90 cps.

The character of the plesimeter impact sound is determined by the nature of the plesimeter. The ivory and wooden plesimeters of an earlier period were shrill and noisy because they were stiff. In digito-digital percussion now used almost exclusively the impact note is less striking yet clearly discernible. It is this plesimeter impact note which is heard predominantly in the dull percussion note and exclusively in the absolutely flat percussion note. According to Selling a plesimeter of soft rubber is almost silent.

Another older method by which the frequency composition of percussion notes was studied many years ago by Cartex (241) is that which involved photographing manometer flames. In this method devised by Koenig (see Miller (1103)) in 1862 the gas pressure to the flame is modulated by a

delicate membrane which vibrates with the sound to be studied. Variations in the height of the flame are what are photographed on moving film to produce a record of the sound vibrations. Selling applied Snodgrass' method to this problem (see Miller (1103)). Oscillographic like tracings were made by a machine which took the vibrations of the gramophone disk and after passage through a system of delicate compound levers marked them on the smoked paper of a kymograph.

Montgomery (1111) states that the fundamental if such it can be called of vesicular (resonant) percussion and of normal vesicular breathing is approximately 108 cps, a close match with 128 cps, the fundamental of man's voice. The fundamental of a woman's voice is closer to 216 cps.

It is a general experience that tympanitic percussion notes are more musical than the resonant ones; their pitch is more easily placed in a musical scale. Selling's string galvanometer recordings of tympanitic notes (137a) displayed more regular vibrations than were found in resonant notes which produced complicated oscillograms. The same finding was made by Witz and Lindemann (reported by Lühr (419)) who used an ingenious but ingenious method. They obtained an artificial membrane by plunging a metal ring into a copoly solution. This delicate membrane vibrated in a highly sensitive manner with sound vibrations and when placed flat it reflected an image on moving photographic film. It is embodied in an oscillogram of the sound.

**THE NATURAL FREQUENCY OF THE THORAX.** It is clear from the previous exposition that the thorax has a natural frequency<sup>1</sup> which is demonstrated by an analysis of percussion notes. The natural frequency in general varies inversely with the volume of the thorax. When a subject sings a low note the hand placed on the chest will feel the vocal vibration; the thorax behaves like a Helmholtz resonator. If the subject sings

<sup>1</sup> Natural specific or intrinsic vibrational frequency depends on the tendency of an elastic structure when deformed from its position of rest or equilibrium to return to that position. The character of the structure's vibration during the period it is returning to its position of rest is determined by its modulus of elasticity; frequency is directly proportional to density and inversely proportional to volume.



an ascending scale, the vocal thrill, palpated on the surface of the chest, will become weaker and disappear. The threshold is higher when the singer is in expiration. The matching of the pitch of the voice to the volume of the thorax is a factor determining whether vocal resonance is present or not. Vocal resonance is the rule in men because of good match. In women vocal resonance is rare because, although the natural frequency of the thorax is higher, the voice is even more shrill than the thorax can match. On the other hand, infants and children are likely to show conspicuous vocal resonance because of having close to ideal match of their more shrill voices to the higher natural frequency of the thorax.

In the strict sense, the thorax is a double coupled system comprising, on one hand, the thoracic wall—particularly the rib cage—and, on the other, the lungs. The natural frequency of the stiffer chest wall is somewhat higher than that of the enclosed structures.

The natural frequency of the thorax is an important factor in the manner in which murmurs are altered in quality in transmission to various parts of the chest. It has been noted that those frequency components of a heart murmur which are in the same general range as the natural frequency of the thorax tend to be transmitted best to various areas of the chest (see p. 150). An experiment comparable with this one of nature is to place a series of tuning forks one after another on the anterior portion of the chest and by palpation gauge the extent to which these vibrations of pure pitch are transmitted to the back. (In actuality satisfactory results from this experiment (1099) are vitiated by circumferential conduction of the vibrations in the rib cage.) Disease processes in the lungs and pleura may have the result that the thorax, or a part thereof, resonates differently from normal. The lower pitched components are diminished in intensity, and one says that the percussion note is higher pitched. This conclusion is due in part to the fact that the more shrill plesimeter impact note is left to impress the auditory perception to a greater extent. To some extent also the higher pitched note is the result of a higher natural frequency of resonance of the diseased thorax. Alterations in this frequency of resonance are important factors in the

genesis of "I to A change" change in breath sounds, and other phenomena discussed below.

### BREATH SOUNDS

The character of the breath sound, as detected by stethoscope or microphone at the surface of the thorax, is determined to a considerable extent by the resonating and sound conducting properties of the lung tissue between the larger airways and the point of detection. The average pitch of normal vesicular breath sounds varies in the same manner as the percussion notes, being progressively lower in infants, adult females, adult males, and patients with emphysema.

Diagnostic changes in breath sounds involve principally the expiratory phase. The stethoscopist should pay particular heed to the character of this phase.

Normal vesicular breath sounds are demonstrated in Figure 472. Expiration is relatively noiseless. Electrical interference at 60 cps is present.

The inspiratory "murmur" or normal vesicular breath sound has its origin at the ileocolic (189) and is the result of turbulence created when air currents spread out into the myriads of air sacs. It is well heard at the surface of the chest because it is generated at close hand. Movement of air in the larger airways is not necessary for its production. One variety of cardiopulmonary murmur is of fundamentally the same origin and has the same character as the vesicular sound (see p. 223). Other evidence for the vesicular origin of the vesicular inspiration sound (note the appropriateness of the terminology) is provided by the auscultation of exenterated lungs (189) and of hernia of the lung (1361). The expiratory sound on the other hand has its origin at bifurcations in the larger airways by a mechanism comparable to the libid pipe of the church organ (1044). Because of the porous sound absorbing intervening lung tissue it is not surprising that the expiratory sound normally should be poorly heard at the surface of the chest.

In the breath sounds recorded from the immediate vicinity of the trachea (Fig. 473) the properties of the thorax are not the major controlling factors. The pitch of the sounds which are actually noise in inspiration and expiration is

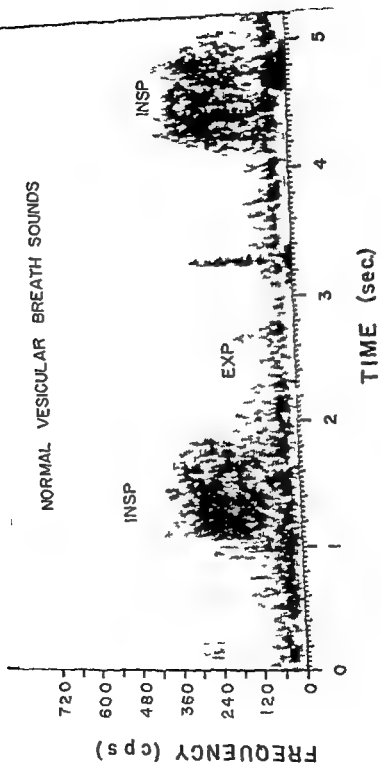


Fig. 3. Normal vesicular breath sounds.

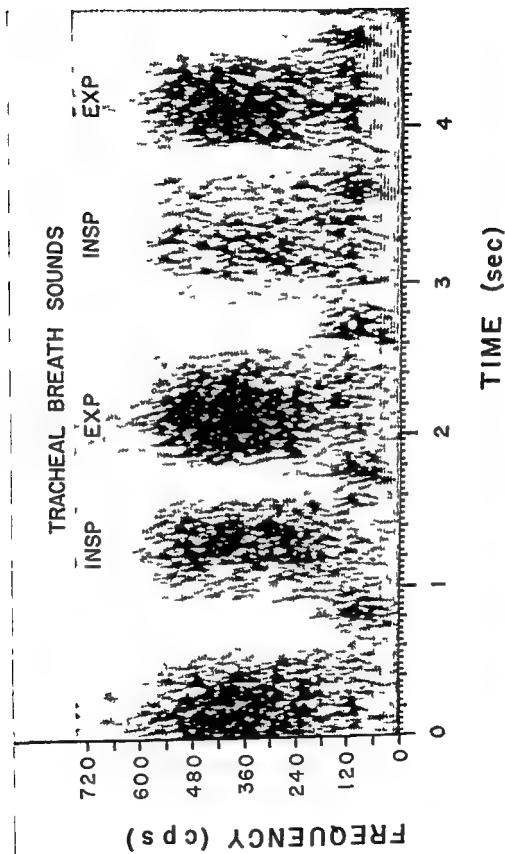


FIG 473 Tracheal breath sounds

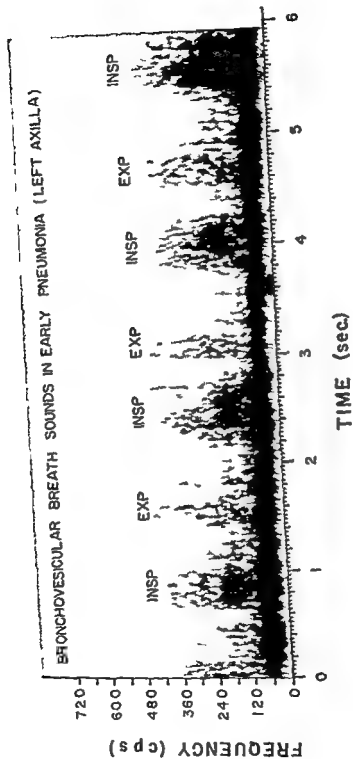


FIG 471 Bronchovesicular breath sounds in early pneumonia (left axilla)

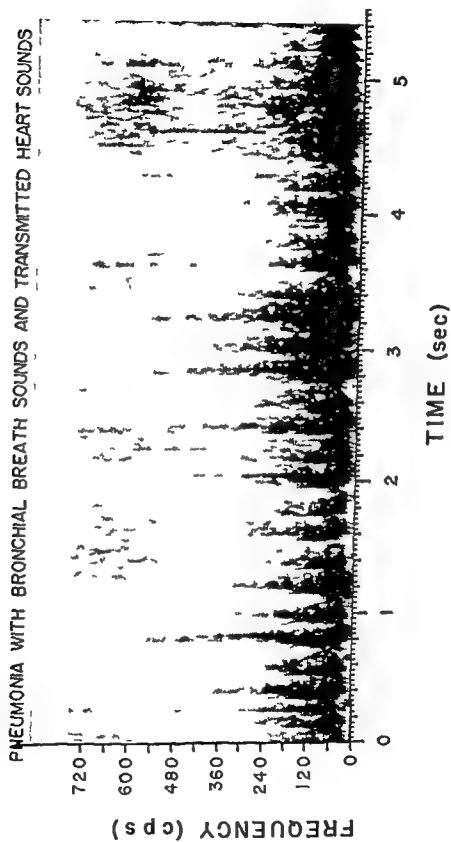


FIG. 175 Bronchial breath sounds (at frequency of 500 cps up) and transmitted heart sounds in case of pneumonia of the left lower lobe

roughly the same but the expiratory sound is appreciably louder. The writer has observed variation in the frequency level of the 'center' of the tracheal breath sound. The variations appear to depend upon the dimensions of the trachea both length and caliber. As might be expected the average pitch of the tracheal sound is lower in persons who appear to have larger trachea and vice versa.

When pneumonia produces changes in the sound-conducting properties of lung tissue expiration which is ordinarily relatively noiseless becomes audible and characteristically bronchovesicular respiration develops (Figure 47). Again electrical interference at 60 cps is present as well as some at 120 and 180 cps. It may be noted that with partial consolidation it is still only the lower pitched portion of the tracheobronchial sound which are heard at the surface of the thorax. At this stage also the vesicular or purratory sound is likely to be diminished which is particularly the case in Figure 47. In many of the alveoli are not functioning because of the condensation.

Further consolidation of pulmonary tissue

such as occurs in later stages of pneumoconiosis pneumonia results in the audibility at the surface of the chest of the vibrations produced in the tracheobronchial tree. In experiments which are now being made Martin and Mueller (101) determined the natural vibrational characteristics of the tracheobronchial tree at several levels (see graphic representation in reference 10). These studies indicate that sound produced by the mechanism of the tubal pipe at the levels of the tracheobronchial tree when the diameter of the tube is 3 to 10 mm in diameter is at a point below the bifurcation of the trachea will have a frequency in excess of 1,000 cycles per second. Consolidated lung tissue because of its better conducting properties permit the detection of these vibrations at the surface of the chest. The result is so-called bronchial or tubular breath sound. These are demonstrated in Figure 47. In the example demonstrated an additional feature of interest is the presence of heart sound confined largely to the frequency range below 240 cps. Because the pneumonia in this case was in the left lower lobe the heart sound was transmitted

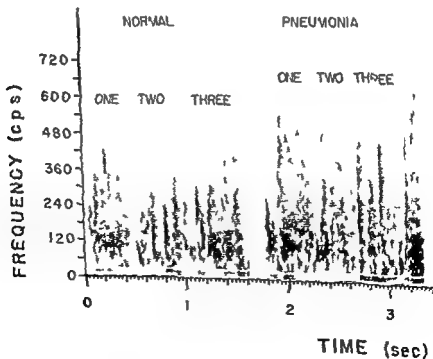


FIG. 46 Augmented whistled sounds in early pneumonia. The change parallels the development of bronchovesicular breath sound. The accentuation involves both intensity and frequency span.

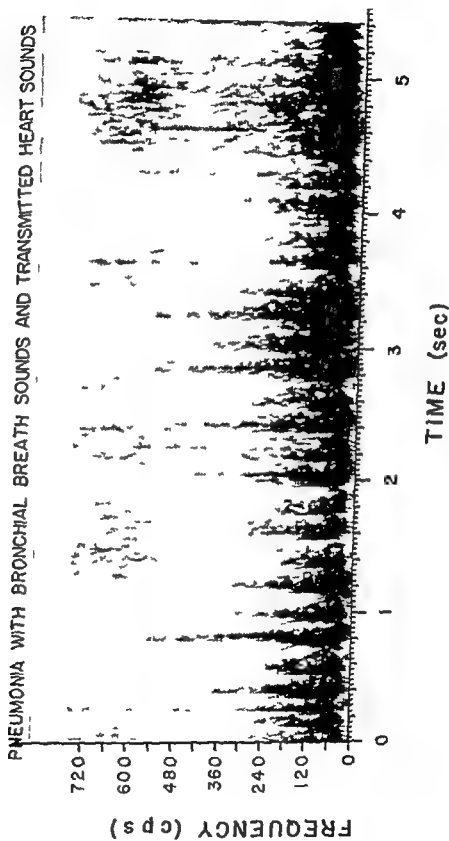


FIG. 47a. Bronchial breath sounds (at frequency of 400 cps up) and transmitted heart sounds in case of pneumonia of the left lower lobe.

to the microphone with abnormal clarity. Pronounced tachycardia was present.

Martini and Mueller (101b) demonstrated in model that pronounced damping of sound vibration occurs in tubes of 2 to 3 mm in diameter. In tubes of 1 mm in diameter very little conduction occurs. This was the basis for their conclusion that the bronchial breath sound have their origin in bronchioles larger than 4 mm in diameter. At this level the bronchiole has a cartilaginous wall and its natural frequency would be expected to be little influenced by the presence of surrounding alveolar consolidation. The appearance of bronchial breathing with the development of consolidation is a matter of transmission of vibrations which occur normally. Anatomic studies revealed that in the adult thorax bronchioles of 2 to 3 mm diameter lie 3 to 4 cm from the surface of the chest and in some areas 5 cm. Near the vertebral column in

the intercostular area, these smaller airways may be as close as 1 to 2 cm to the surface. These measurements are an indication of the depth of consolidation which is necessary for the development of bronchial breathing.

Lung Helmholtz resonators. Martini and Mueller (101b) demonstrated components of approximately 1000 cps in bronchial breath sound. In the experience of Cabot and Dodge (205), the highest frequencies lay in the range of 600 to 1000 cps.

#### WHISPERED AND SPOKEN SOUNDS

Corresponding to the presence of bronchovesicular breath sounds the *whispered voice sounds* may be exaggerated in intensity and frequency span, as is illustrated in Figure 47b.

When bronchial breath sound are present the whispered sound (95b) takes on the character which gives the phenomenon the name whispered

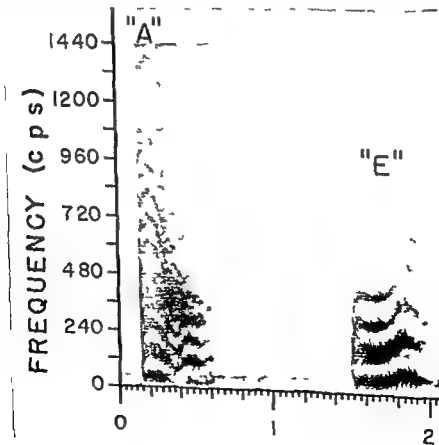


FIG. 48 Spectrograms of sound I and A spoken directly into microphone.



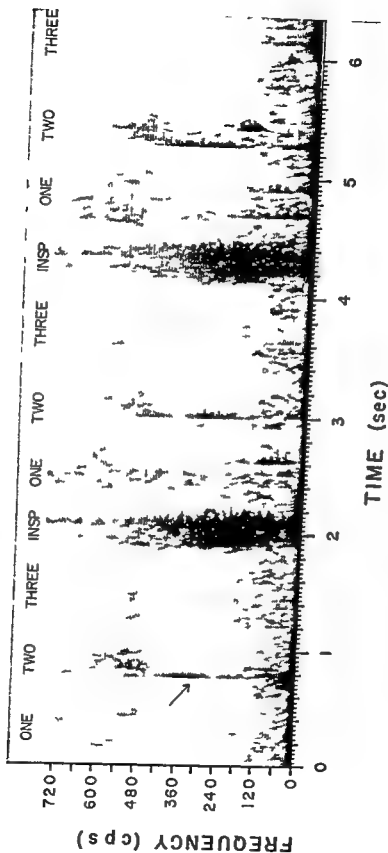


FIG 477 When pered pectoriloquy changes paralleling the development of bronchial breath sounds in pneumonia. Note that the sounds have the same general frequency range as the breath sounds in bronchial breath sounds (470). Note also the sharp plosive sound of two (a so called plosive) indicated by the arrow.

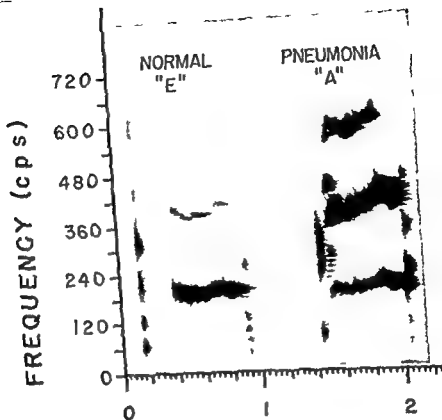


FIG. 480. I to A change over consolidated lung

is I or an (one two three) The word for one was pronounced E in his province hence the origin of the I to A sign.

Froehel and Stockert (196) found that one vowel sound might be transformed into another with no particular predictability.

The patient from whom the recording analyzed in Figure 481 was made had a pleural effusion on the right. The alteration is somewhat different from that seen in Figures 479 and 480. Here A when detected at the upper level of the fluid sounded to the ear something like A (as in cat) with a bleating quality typical of egophony. The physical changes produced on the side of the fluid are (1) marked attenuation of the fundamental (at 160 cps), (2) reinforcement of the second harmonic (at 320 cps) and (3) attenuation of the fourth harmonic (at 640 cps). The behavior of the hemithorax which is partially filled with fluid is that of a muffler. A band filter, it has a

higher center frequency and narrower frequency pass band.

In the past much of speculative nature has been written about the physical origin of egophony. Norris and Linds (1147) surveyed the theories in this manner:

- (a) Flattening of noncartilaginous bronchi by pressure of the effusion so that they act like reed (Lancet).
- (b) vibrations of the walls of small bronchi as actual collision—interruption of air currents (Wintrich).
- (c) the articulation of vibration—only the higher harmonics passing through the effusion (Stone).
- (d) vibrations passing through a thin layer of fluid.

Obviously studies by sound spectrography lend support principally to the theory of Stone.

Dunn and colleagues (188) studied the acoustic properties of the normal and abnormal chest using a phonation method essentially identical to that discussed in this section. The intensity of elements in various frequency bands at various

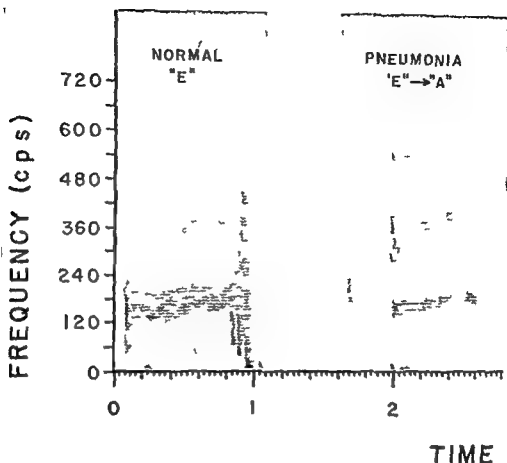


FIG. 479.  $\Gamma$  to A change over lung consolidated by pneumonia

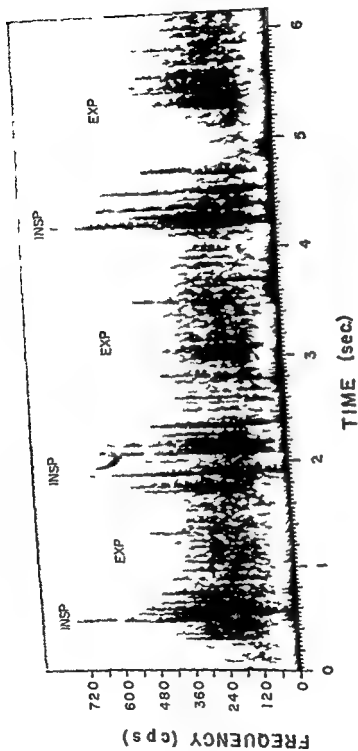
pectoriloquy (Fig. 477) the sounds are high pitched with the same general frequency range as the inspiratory and expiratory sounds in bronchial breathing. (Different cases are illustrated in Figures 475 and 477) The genesis and significance of bronchial breath sounds and whispered pectoriloquy are identical. It is of interest to note in Figure 477 that the sharp "t" sound of "two" (also called "plosive") is evident each time as a circumscribed sound indicated in the first instance by an arrow.

Analysis of the alterations in the *spoken vocal sounds* has yielded some of the most interesting results of the present studies. Contrary to what at first thought might seem to be the case, E' is, on the average, a lower pitched sound than A'. The analyses of the sounds A' and  $\Gamma$ ' presented in Figure 478 were made from recordings of the sounds spoken directly into the microphone. Both sounds have conspicuous harmonics with characteristic curvature. In sound A' the frequency span is greater, and the most intense

(loudest) harmonics are in the vicinity of 360 cps as compared with 150 cps in the sound  $\Gamma$ .

Over consolidated lung tissue E' spoken by the patient is likely to sound like A'—the so called  $\Gamma$  to A change. The analyses displayed in Figures 479 and 480 demonstrate that this change is due to selective reinforcement (or at least selective increase in transmission) of certain ones of the higher harmonics of  $\Gamma$  and attenuation of lower ones. The result is a sound in which the harmonic pattern resembles more that of A' than that of the parent sound  $\Gamma$ .

This type of physical sign was probably discovered simultaneously about 1922 by Shibley in China (1385) and Froehlich and Stockert in Vienna (496). Shibley reported that the five vowel sounds A, E, I, O, U, all become A or Ah in a localized area over fluid or consolidation and indeed also over a large gasser A medical missionary in China, he came upon this sign rather accidentally from having his Chinese patients



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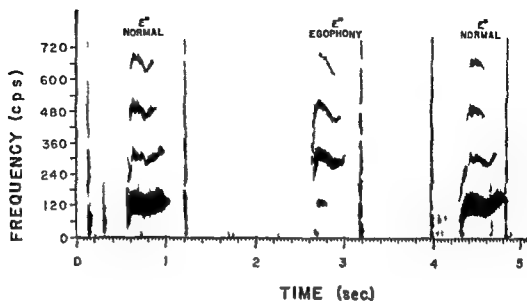


FIG. 481. Alteration in  $I$  at upper level of pleural effusion.  $I$  sounded like  $A$  and had the bleating quality of egophony.

loci was determined. As might be expected the method gave results which were the same as those reported here. However obviously the method was much more arduous and its final product less graphic.

#### RULES

Rules, with the exception of the musical sibilant ones produced in the bronchioles and occurring most dramatically in bronchial asthma, are circumscribed noises of limited duration but considerable frequency span. It is possible by the method of sound spectrography to provide a physical definition for the extensive descriptive terminology for rules. Two extreme examples are illustrated in Figures 482 and 483.

The patient illustrated in Figure 482 had extensive bronchiectasis with superimposed pneumonia. Numerous moist, bubbling rules are present in both inspiration and expiration. The breath sounds are bronchovesicular in character. In the second 'inspiration' a wing-shaped harmonic at approximately 600 cps indicates a slight bronchiolar squeak which occurred at that point. Most of the rules in this instance display a fine harmonic pattern.

By way of contrast, the patient illustrated in Figure 483 had dry crackling rules limited to expiration. Pulmonary tuberculosis was present with predominant involvement of the right apex, from which region this recording was made. The harsh character of the inspiratory sound is evident. As to the rules, these sounds are characterized by a more diffuse uninterrupted frequency distribution than in the moist rules illustrated in Figure 483. From other experience it is known that sharp sounds with this pattern are to the ear snapping and crackling in quality. In studying cardiovascular sound for example it is found that systolic clicks and the opening snap of mitral stenosis have this same general appearance in the spectrogram.

The musicality of the rules in bronchial asthma is the result of the presence of conspicuous harmonics (see Fig. 484 also Fig. 41B). Expiration is prolonged and occupied by harmonics as demonstrated in the illustrations. Close scrutiny of the tracings reveals, as might be expected, that more than one musical instrument has been recorded. One produces a fundamental at 240 and a second harmonic at 480 cycles per second. This same sound generator also produces a further sound in

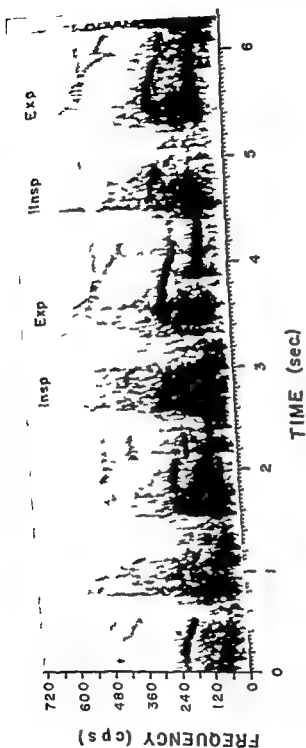
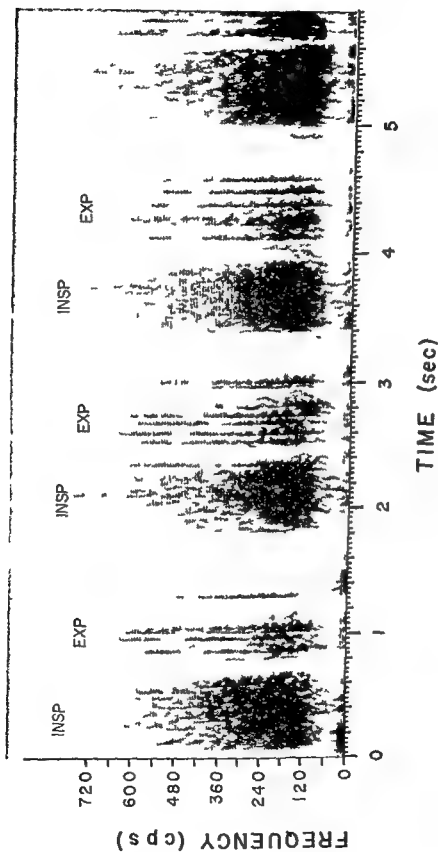


FIG. 18. Musical notes of the HVC sounds. The notes are much longer.



1 to 483 Dry crackling, rales in expiration. Note the difficulty in differentiating dry rales from pleural friction rub (cf Fig 485)

## SECTION VIII

*Miscellaneous Sonic Phenomena of Medical Interest*



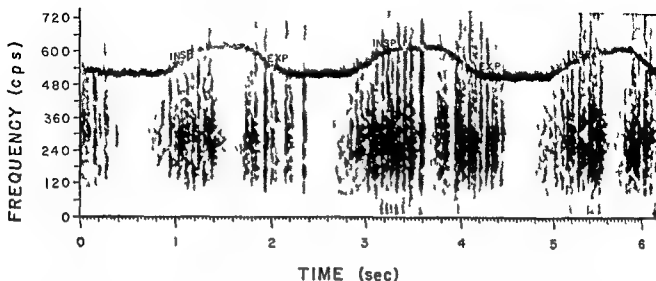


FIG 485 Pleural friction rub in L R (176906) 49 year old patient who demonstrated a pleural rub of several weeks following an automobile accident with rib fractures. The rub consists of a series of transients in rapid succession.

inspiration. In two cycles, late in expiration a pair of harmonics are seen at 180 and 360 cps. The two harmonics crossing at 480 cps almost certainly were generated in different bronchioles.

#### PLEURAL FRICTION RUB

The mechanism of the pleural friction rub is "stick and slip" (p 138) as is that of the pericardial friction rub. At one extreme stick and slip may be so evident that discrete sounds, transients, are evident to the ear and are demonstrated in the sound spectrogram (Fig 485). This

coarse type of pleural friction rub may be difficult to differentiate from coarse, dry rales. At the other extreme the pleural friction rub may be difficult to distinguish from abnormal breath sounds, specifically the exaggerated breath sounds of partial pulmonary consolidation. (This is a situation analogous to the confusion of some periodic frictions for endocardial murmurs.) In such instances stick and slip probably is operating independently in innumerable local zones. Although in each area a faint transient is produced, in the aggregate the result is a murmur like sound.

## CHAPTER 25

# Miscellaneous Sonic Phenomena of Medical Interest

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**BOWEL SOUNDS** The experienced abdominal surgeon is able to extract much information about intestinal motility or obstruction from the character of the bowel sound. Several authors, beginning at least with Walter B. Cannon in 1903 (257) have studied abdominal sounds (390-965). Some (453) have used these sounds for an objective measure of the effects of antiperistaltic drugs. A difficulty of this method is introduced by the fact that the stomach and colon dominate in the production of noises, overshadowing the small intestine which is most sensitive to antiperistaltic drugs. The technique and possibly the research value of a systematic review of the subject of bowel sounds has not yet been explored.

The study of joint sounds is in a similarly undeveloped state.

Lian and Odinet (933) observed that in the presence of a celiac perforation in one iliac fossa

and in cultation (or recording) in the other resulted in the detection of a double sound as opposed to the single sound produced in the normal.

Peltier (1198B) describes a sound conduction test for fracture of the femur and dislocation of the hip. The method would appear to have particular usefulness under circumstances of emergency. With the patient supine a methoscopa is placed firmly on the symphysis pubis and each patella is struck lightly with a finger. A clear, distinct sound is transmitted by the unbroken bone column on the normal side, whereas a softer, less distinct sound is heard from the injured side. The progress of healing can be nicely followed by means of this test. Bone cysts and tumors and possibly effusion of the hip joint may produce change. Modification of the test should be applicable to fractures in other bones.



## SECTION IX

### *Technical Appendix*



# TECHNICAL APPENDIX

*Prepared with the Assistance of Mr George A Webb*

## SPECTRAL PHONOCARDIOGRAPHY

Discussion of the problems of instrumentation for spectral phonocardiography can be divided into the following major parts: first the detection of the sound on the chest wall; second the storage of these sound along with the correlative physiological data; third analysis of the sound for tonal quality; and fourth the final written record (1014).

The equipment must be considered till in the developmental stage. Various parts of it are being used duly in clinical studies while the development advances. All basic operational functions discussed have been tested and used but one unified easily operated let alone commercially feasible instrument has not yet evolved.

**DETECTION OF SOUND** Most heart sound transducer can be placed in one of three classes: (1) displacement where no contact is made to the chest wall (e.g. Groom's microphone (610)); (2) pressure where an air chamber between the chest wall and a diaphragm converts the wall displacement to a pressure (most phonocardiography is done with transducers of this type); and (3) contact where the chest wall drives a sensing element directly. See p 503-504 for a more detailed discussion of microphones.

Respiratory variations are usually eliminated by having the transducer ride the gross movement of the chest wall with breathing. The low frequency bulk-tic components artifacts for purposes of phonocardiography are attenuated by acoustic filtration inherent in the physical design of the transducers and/or by electrical filtering. It is important that the undesirable low frequency pressures present at the microphone position do not overload the system and thus generate new high frequency components which have no existence in physiological fact.

Several studies (1031) have indicated that the amplitude of the frequency components of a given heart sound decreases toward the higher frequencies at the rate of about 16 db per octave. The ear at low level of pressure approaching the threshold of audibility, has a sensitivity curve which rises at the rate of about 12 db per octave. In many respects the feature of the auditory mechanism is ideal for an audition. The 16-db per-octave slope of the heart sounds suggests that a phonocardiographic system which has a sensitivity of about 12 db per octave may be desirable. When the full tonal quality of the sound is to be illustrated, low frequency attenuation should be used to the extent that the highest and lowest amplitudes at the extreme frequency limits come within the amplitude dynamic range of the final write-out. Care must be exercised in this regard since during the heart cycle the dynamic range may be great at any given frequency level. The range again is beyond the scope of the write-out and can be best handled by logarithmic amplification after analysis.

**REPRESENTATION** Storage of or displaying the full tonal quality of the sound in one form of write-out representing many frequency components is required. Either multiple simultaneously operating analyzers—such as Munnheimer (1031) Weiss and Weber (1001) and others—are required or alternatively storage and repeated analysis of the same sound. We have used the second method which for the information provided seems to have the economic advantage at the present time.

All data are first recorded on magnetic tape. Selected portions are transferred to magnetic loop or magnetic drum for repeated playback and analysis.

The equipment first used to establish the



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**RECORDATION STORAGE.** For displaying the full tonal quality of the sound, some form of write-out representing many frequency components is required. Either multiple simultaneous operating analyzers—such as Munshower (1011), Mraz and Weber (1001) and others used—or required or alternatively storage and repeated analysis of the same sounds. We have used the second method which for the information provided seems to have the economic advantage at the present time.

All data are first recorded on magnetic tape. Selected portions are transferred to magnetic loop or magnetic drum for repeated playback and analysis.

The equipment first used to establish the





not important. Most commercial FM demodulators are designed to drive galvanometers which is not required in this voltage-operating system.

One of the open questions in the final design of a spectral phonocardiograph concerns the best method of repetitively playing back the recorded information for the multiple analysis. The magnetic drum has the disadvantage of a fixed play time and from our experience a difficulty in attaining low wow and flutter when an 8-to-1 speed change is required. When the recording head rides on the drum excessive wear results although this may be an acceptable expedient. If the heads are picked away from the drum is a common practice in computer work a very large drum is required to accommodate the frequency response required. For using tape loops both rotating head and moving tape with stationary head have been developed. Whether or not separate transport are to be used and whether all original recordings are to be preserved are considerations to be weighed in choosing a loop play back.

**ANALYSIS.** The first rudimentary filters for analysis of heart sound were the bell and diaphragm of the stethoscope. These provided a choice between two rather broad band pass filters which aided in focusing on one or another aspect of the sound. When amplitude vs. time recordings (conventional phonocardiograms) were made it was evident that some form of low frequency attenuation was required. Methods vary from a simple 6 or 12-db per octave attenuation across the entire audible region to very sharp low-cut filters to demonstrate the components above 1000 cycles (as used by Rodbard (1293)). Another approach was to pass the sounds simultaneously through multiple filters making multiple amplitude vs. time recordings. There is obviously an economic limit to the number of such channels.

The analyzing method on which spectral phonocardiography is based was that developed by the Bell Telephone Laboratories (1247, 1248), and called visible speech. In this system in order to obtain a maximum of information the abscissa remains time but the ordinate becomes frequency where intensity of the sound at a

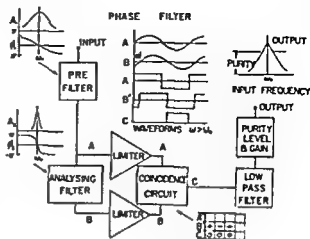


FIG. 484. Schematic of phase filter.

given time and frequency becomes a step on the gray scale.

The present method is to examine many times the group of heart sounds to be analyzed. The rate of examining is eight times the speed at which the original recording was made. This speedup decreases the time required for analysis and allows the use of convenient sizes of filter components.

The analyzing filter operates at a fixed frequency of 15 kilocycles per second. The sounds to be analyzed are heterodyned with a local oscillator so that the lower side band passes through the filter. The frequency of the oscillator is coordinated with whatever medium it changes the vertical writing rate of the write-out. The output of the filter is used to intensify the modulate the write-out.

Two types of filters are presently available. First is a conventional two-stage LC tuned filter. As explained on p. 89, the five filter systems we tested are arbitrarily designated A through E. A represents a relatively high Q where the filter rings and is quite frequency sensitive with little time information. F represents the other extreme where the Q is low and much frequency information is lost. Filter C represents a compromise in which some degree of both timing and frequency detail is retained. We have arbitrarily chosen filter C as the one having the best timed and tonal characteristics for cardiovascular sound.

Figure 488 shows the basic operation of the second type of filter—the phase filter (722, 1101).



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The analyzing method on which spectral phonocardiography is based was that developed by the Bell Telephone Laboratories (1247, 1248), and called variable speech. In this system in order to obtain a maximum of information the absolute remaining time but the ordinate becomes frequency whereas intensity of the sound at a

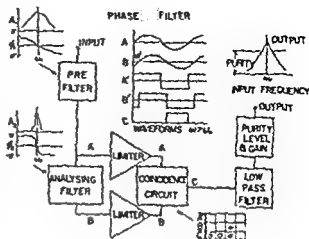


Fig. 4-8. Scheme of phase filter.

given time and frequency becomes a step on the graph scale.

The present method is to scan many times the group of heart sound to be analyzed. The rate of scanning is eight times the speed at which the original recording was made. This deceleration decreases the time required for analysis and allows the use of convenient sizes of filter components.

The analyzing filter operates at a fixed frequency of 15 kilocycles per second. The sounds to be analyzed are heterodyned with a local oscillator so that the lower side band passes through the filter. The frequency of the oscillator is coordinated with whatever mechanical changes the vertical writing rate of the write-out. The output of the filter is used to attenuate its modulation.

Two types of filters are presently available. First is a conventional tunable LC tuned filter. As explained on p. 89, the five filter systems we tested are arbitrarily designated A through E. A represents a relatively high Q where the filter rings and is quite frequency selective with little time information. F represents the other extreme where the Q is low and much frequency information is lost. Filter C represents a compromise in which some degree of both tuning and frequency detail is retained. We have arbitrarily chosen filter E as the one having the best tuned and tonal characteristics for cardiovascular sound.

Figure 4-8 shows the basic operation of the second type of filter, the phase filter (722, 1101).



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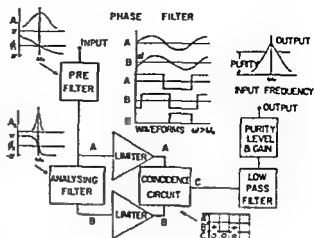


FIG. 458. Schema of phase filter.

given time and frequency becomes a step on the gray scale.

The present method is to scan many times the group of heart sound to be analyzed. The rate of scanning is eight times the speed at which the original recording was made. This speedup decreases the time required for analysis and allows the use of convenient sizes of filter component.

The analyzing filter operates at a fixed frequency of 15 kilocycles per second. The sounds to be analyzed are heterodyned with a local oscillator so that the lower side bands pass through the filter. The frequency of the oscillator is coordinated with whatever mechanism changes the vertical writing rate of the write-out. The output of the filter is used to intensity modulate the write-out.

Two types of filters are presently available. First is a conventional two-stage LC tuned filter. As explained on p. 89, the five filter systems we tested are arbitrarily designated A through E. A represents a relatively high Q where the filter rings and is quite frequency selective with little time information. F represents the other extreme where the Q is low and much frequency information is lost. Filter C represents a compromise in which some degree of both timing and frequency detail is retained. We have arbitrarily chosen filter C as the one having the best timing and tonal characteristics for cardiovascular sound.

Figure 458 shows the basic operation of the second type of filter, the phase filter (722-1101).

The purpose of filter A is primarily that of removing the upper side band. It provides very little filtering by itself. The analyzing filter (A2) is the same filter as that used in the system referred to in the last paragraph. However, instead of detecting the amplitudes from the filters, the two signals are passed through the two limiters, providing square waves at the points A and B. A time difference between the zero crossings of signals A and B exists, depending upon the relationship of the frequency of the input signal to the center frequency of the analyzing filter. The coincidence circuit produces an output pulse only for the period that both A and B are negative. The low pass filter takes the average value of this coincidence pulse. The purity level is a clamp circuit control which in effect removes that portion of the response curve below the corresponding purity level and thus narrows the bandwidth. This narrowing of bandwidth has been accomplished without introducing further ringing, such as occurred with the first filter. The result of this type of filtering, illustrated in Figure 44, is to accentuate continuous tones over random noise. Where the time frequency characteristic of a musical murmur is more important than the actual intensity of the musical tone, the second system is superior.

**THE WRITE OUT** In the original equipment a magnetic disk like drum twelve inches in diameter and one half inch wide and a writing cylinder like drum four inches in diameter and eight inches long are mounted on the same axis. The electro-sensitive paper is wrapped around the writing drum. As the drums rotate during analysis a marked stylus moves up the drum, changes the frequency of the local oscillator during each rotation and writes the filter output on the record drum. The principal disadvantages of this method are lack of precise intensity control in marking the paper, a short gray scale range, and the ease of smudging the record. An attempt to overcome some of these difficulties was made by using photographic paper on a second drum operating on the same axis but in a light tight box above the original drum (Fig 43). The difficulty of manipulating the photosensitive paper under these circumstances shelved this approach.

Figure 489 presents a simplified block diagram

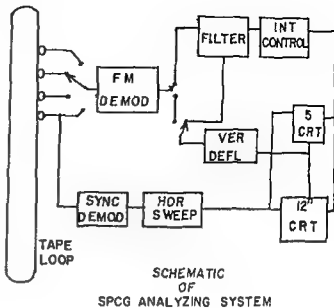


FIG 489 Schema of SPCG system

of what, at the present writing, seems to be the most satisfactory system. A cathode ray tube is used for the final display with a camera making a photographic record. The unique feature of this system is the ability to combine two dimensional linear deflection pulse intensity information the same as in a TV raster, with the conventional oscillographic display.

Either a loop or a drum storage mechanism is used for repetitive playback. Two mixed channels may now be used on the drum and four channels on the loop. The horizontal sweep of the 12" monitor tube and the 5" read out tube is linear and synchronized from the loop. Unlike the mechanical system where the magnetic drum and write out mechanism are locked together, the reproducibility here is dependent upon low jitter of the sync pulse uniformity of tape speed, and linearity of sweep. The main advantage here is that the analyzed portion can be chosen to be all or part of the loop depending upon sweep speed and sweep delay. The vertical deflection of the two CRT tubes can be either a linear sweep synchronized with the frequency change of the filter local oscillator or it can be switched to the demodulated signals from one of the recorded channels to provide an oscillogram.

The sweep times involved in the raster scan are of the order of 600 milliseconds maximum for the horizontal sweep and five minutes for the vertical during the total analysis. The slow sweep

rates place unusual requirements upon the intensity control circuit. The cathode-ray tubes commonly used are magnetically deflected and electrostatically focused. The beam is gated by the cathode and the grid is used for intensity modulation. For 10 KV accelerating potential and the sweep speed a bias of 4.5 volt is required with a modulation signal of 2 volt peak. Since screen burning occurs very soon after the 2 volt level is reached careful limiting circuit are required. The problem of grain lies with the cathode-ray tube and not with the film. What is needed is a homogeneous phosphor.

The relation ship between grid drive and light intensity is not linear so some form of gamma correction is necessary. A rooster circuit (11-13) is included to compensate for this effect. This in addition to any logarithmic compression that may be introduced in the filter circuit to compress the great amplitude range of the sound into the film latitude. With the present equipment about 43 db of signal to the rooster circuit carries the beam intensity zero, the exposure range of the Polaroid film used in making the permanent record.

**TYPE DESIGN CONSIDERATIONS** For wide spread use of the spectral phonoscaphograph a simplified form of information storage and reproduction playback is desirable for economic reasons. For the sake of reduced size and decreased heat output more use of transistors is indicated. Preliminary design and testing have been made on logarithmic compression deflection circuit and power supplies in transistors. For instance the deflection circuit are inefficient although high impedance tubes are used. The cathode ray tubes using the new transparent phosphor under development by several manufacturers would improve the uniformity and resolution of the final display. More basic research into the adequate display of the tonal characteristics of the impact sound (heart sound) is required.

#### OTHER SELECTED TOPICS IN THE TECHNICAL ASPECTS OF THROMBOCARDIOGRAPHY

##### Microphones

The detection of heart sound on the chest wall is usually accomplished by allowing the

movement of the wall to produce pressure changes in a closed volume (the second general type of method mentioned on p. 400). A pressure transducer is connected to this volume. For the tethoscope the cut is the transducer. The bore length and elastic properties of the tubing can affect the results obtained by the tethoscope (see pp. 68 to 70). When microphones are used as the pressure transducer a minimum volume of air is introduced between the chest wall and the diaphragm of the transducer tubing effect can usually be ignored.

For quantitative reproducible results several aspects of heart sound detection need consideration. The pressure which the microphone exerts on the chest wall affects the tension of the surface skin and produces compression of the subcutaneous tissues. As a result sound transmission is affected in a manner which at this time has not been quantified. The variability of these effects between patients is also unexplored. Care must be taken that the large displacement of low frequency sound does not produce pressures in the small enclosed coupling volume of the microphone which is to distort the electrical output. A microphone should be chosen which can either accept the high pressures without distortion or one which has an acoustic filter to reduce their amplitude.

The capacitor microphone depends for its operation on variation of the electrical capacitance between a movable diaphragm and a fixed plate.

One form (610) uses the chest wall or a light aluminum foil on the chest wall as the movable plate. The microphone detect chest wall movement with a minimum of loading of the wall. In addition since no diaphragm sensitive to air pressure is used the pick up of ambient room noise is greatly reduced. The sensitivity of this microphone is dependent upon the basic separation of the two plates which would be of the order of a few thousandths of an inch. Although the adjustment presents problems the above mentioned advantages are important. There is no opportunity for acoustic filtering in this unit.

The second form of the capacitor microphone (684) uses a stiff plastic diaphragm about the size of a dime. The facts that acoustic filtering can be used that it can withstand high pressures with



minimum of distortion<sup>1</sup> and that it is made of inert glass, gold, stainless steel and Mycalex recommend this unit. For the purpose of detecting change in the capacitance a charging voltage of about 200 volts is used. The usable signal is a small variation of this charging voltage (of the order of a few thousandths of a volt). High humidity can be a problem because of electrical leakage in the associated amplifying equipment which necessarily have a high impedance.

The crystal microphone has been quite widely used for phonocardiography (1031). In this transducer the movements of a diaphragm transmit a force to a crystal which generates a voltage when stressed. The principle advantage is low cost. The disadvantages are distortion with high acoustic loading, deterioration with prolonged exposure to either high or low humidity, and complete destruction by temperatures over 120° F. Although some (1031) have used this for exhibited work, its chief utility comes in providing a record of the heart sounds in situations in which wave form is not the first consideration and temporal correlation with other types of records, e.g., the ballistocardiogram, is the main requirement.

The electrodynamic or moving coil microphone (88) consists of a coil mounted on a diaphragm and a permanent magnet. The coil is caused to move in the magnetic field by the vibrations of the diaphragm. A complex acoustic filter is required to insure that the voltage output is proportional to the pressure on the diaphragm rather than to the velocity of the diaphragm coil combination. In the past the large size and weight of this type of microphone and its susceptibility to pick up of hum has impeded wide acceptance in phonocardiography; however, there are some reports of its use (1033).

Dunn and Rühm (385) described a pistonphone which could be used for exhibiting microphones. Other uses are suggested by the statement of these workers that it is a stable and reliable source of pure low frequency tones for general applications in the acoustical fields<sup>2</sup> that is, it can be used as

a sound generator for studies of sound transmission in the body.

A new type of microphone which may contribute to our understanding of the meaning of the heart sounds is the *phono catheter*. This consists of a pressure transducer placed at the end of a catheter to measure the transient pressure conditions inside the heart chambers and the great vessels. Since it is the pressure transients that are of interest it is important that the catheter should not be sensitive to bending and impacts. Careful evaluation of this pick up is needed before the results of its use can be fully accepted. If proved satisfactory much new information can be obtained by this instrument.

### Low Frequency Attenuation

As previously stated, attenuation of low frequency signals is necessary in phonocardiography for two reasons. Signals below the audible region should be attenuated sufficiently to cause no overloading in microphone electronics or display in an oscillogram. Because of the great amplitude range of the heart sounds in the audible region the low frequency component which have the greatest amplitude need to be attenuated to bring them to a level comparable to the high frequency components which are used for timing purposes.

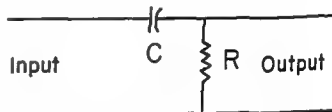


FIG. 490 Circuit for low frequency attenuation

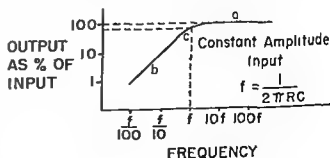


FIG. 491 Output of circuit of type shown in Figure 490

<sup>1</sup> In the condenser microphone there is still risk that strong impacts may overload the microphone and introduce click-like sound which in fact do not exist. The mid systolic click recorded at ILSB in cases of ASD (see Fig. 339) may be a case in point.

(in the case of the heart sound) and for tonal analysis (in the case of murmur).

Low frequency attenuation in phonocardiography is accomplished by means of a circuit of the type schematized in Figure 490. The electrical circuit characteristic of a capacitor (reactance) is symbolized as  $X_C$  and is defined by the formula  $\frac{1}{2\pi f C}$

in which  $f$  is frequency and  $C$  is capacitance. Capacitance is directly proportional to the area of the plates and inversely proportional to the distance between them ( $C = K \frac{1}{d}$ ). Reactance is

frequency-dependent. The characteristics of the entire circuit is dependent on the relationship between  $X_C$  and the resistance ( $R$ ). The unitage for both values is ohm. When  $X_C \ll R$  i.e. is much less than  $R$  (by a factor of 100 or more) then the input about equals the output.

Because of a vectorial relationship between  $X_C$  and  $R$  there is a phase shift at low frequencies. With an input signal which is of constant intensity over a range of frequency up to 2000 cps, output will match input at the upper end of the frequency scale (line A of Fig. 491) and there will be no phase shift. At low frequencies there will be a linear relationship (line B in Fig. 491) between frequency and output with however a 90° phase shift. In this range the attenuation is 6 db per octave or 20 db per decade. At intermediate level of frequency when  $X_C$  has a value between 10% and  $R$  10 there is a curvilinear relationship (line C in Fig. 491) this being with respect both to amplitude of response and to phase shift.

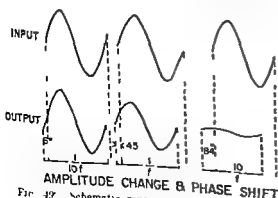


FIG. 49 Schematic representation of phase shift accompanying low frequency attenuation by circuit of type diagrammed in Figure 490.

the transition zone between the two areas of linear response. See Figure 492 for schematic representation of phase shift and amplitude attenuation.

Acoustic attenuation of low frequency components of sound as was practiced by Enthoven (Fig. 1) and many since him has the same characteristics as those indicated in Figures 491 and 492 and the circuit represented in Figure 490 is the electric analog of simple acoustic attenuation.

It is unlikely that the inevitable phase shift which accompanies low frequency attenuation introduces serious error into phonocardiography. Any errors are in the timing of cardiac events and it is unlikely that these will exceed the 0.01 sec limit which is the order of exactitude in

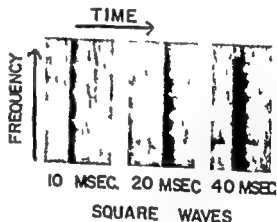


FIG. 493 Artificially splitting of square wave.

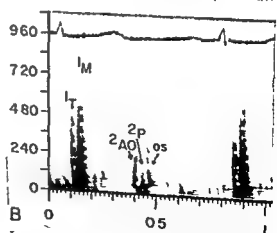


FIG. 494 Artificially splitting of tricuspid closure sound (T) and mitral closure sound (M) when sounds at apex in mitral stenosis stretch out on time scale and played into spectrograph with high gain.

minimum of distortion<sup>1</sup> and that it is made of inert glass, gold, stainless steel and Mycalex recommend this unit for the purpose of detecting change in the capacitance of a charging voltage of about 200 volts is used. The usable signal is a small variation of this charging voltage (of the order of a few thousandths of a volt). High humidity can be a problem because of electric leakage in the associated amplifying equipment which necessarily have a high impedance.

The crystal microphone has been quite widely used for phonocardiography (1031). In this transducer the movements of a diaphragm transmit a force to a crystal which generates a voltage when stressed. The principle advantage is low cost. The disadvantages are distortion with high acoustic loading, deterioration with prolonged exposure to either high or low humidity, and complete destruction by temperatures over 120° F. Although some (1031) have used this for calibrated work, its chief utility comes in providing a record of the heart sounds in situations in which wave form is not the first consideration and temporal correlation with other types of records, e.g., the bull's cardiogram, is the main requirement.

The *electrodynamic* or *moving coil* microphone (88) consists of a coil mounted on a diaphragm and a permanent magnet. The coil is caused to move in the magnetic field by the vibrations of the diaphragm. A complex acoustic filter is required to insure that the voltage output is proportional to the pressure on the diaphragm rather than to the velocity of the diaphragm-coil combination. In the past the large size and weight of this type of microphone and its susceptibility to pick up of hum has impeded wide acceptance in phonocardiography; however, there are some reports of its use (1033).

Dunn and Rubin (385) described a *phonophone* which could be used for calibrating microphones. Other uses are suggested by the statement of these workers that it is a stable and reliable source of pure low frequency tones for general applications in the acoustic field<sup>2</sup>; that is, it can be used as

a sound generator for studies of sound transmission in the body.

A new type of microphone which may contribute to our understanding of the meaning of the heart sounds is the *phono catheter*. This consists of a pressure transducer placed at the end of a catheter to measure the transient pressure conditions inside the heart chambers and the great vessels. Since it is the pressure transient that is of interest it is important that the catheter should not be sensitive to bending and impacts. Careful evaluation of this pick up is needed before the results of its use can be fully accepted. If proved satisfactory, much new information can be obtained by this instrument.

### Low Frequency Attenuation

As previously stated, attenuation of low frequency signals is necessary in phonocardiography for two reasons. Signals below the audible region should be attenuated sufficiently to cause no overloading in microphone, electronic or display in an oscilloscope. Because of the great amplitude range of the heart sounds in the audible region the low frequency component which have the greatest amplitude need to be attenuated to bring them to a level comparable to the high frequency components which are used for timing purpose.

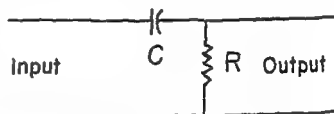


Fig. 400 Circuit for low frequency attenuation

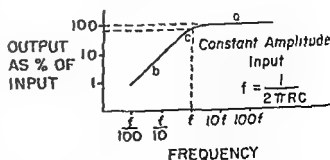


Fig. 401 Output of circuit of type shown in Figure

<sup>1</sup> In the condenser microphone there is still risk that strong impacts may overload the microphone and introduce click-like sound which in fact do not exist. The mid-voltage click recorded at 115 B in case of ASD (see Fig. 319) may be a clue in point.

## References

1. ARNOT M F. Correlation of the aortic arch with the aortic valve. A statistical study and histological report of 200 recent deaths with autopsies of ten to 100 years of age of the aortic arch in respect to the age of 2 years. *Am Heart J* 3: 299 and 3: 1928.
2. ABBOTT M J. On the incidence of bacterial inflammation process in cardiovascular defect and on malformed embryonic. *Ann Clin Med* 14: 180 1920-9.
3. ABRAHAM D C AND WILKINSON I. Pulmonary stenosis with normal aortic root. *Brit Heart J* 13: 19 1951.
4. ALEXANDER AND CARRALL R. The diagnosis of the pulmonary disease. *Am Heart J* 24: 314 1927.
5. ALEXANDER F. The urethral fistula in congenitally active circulation. *J Appl Physiol* 24: 171 1923.
6. ALLEN A. Verdrückte Aortenklappen. *Ztschr Kreislaufforsch* 10: 313 1927.
7. ALLEN J C I AND HILLON R. Aortic fistula stem with malformation of the age of 3. *Brit Heart J* 18: 170 1956.
8. ALLEN R. L. AND KENNEDY C. M. D. WILKINSON I. J. B. NOLAN J. KENNEDY J. AND CARRALL R. Report on the motion and in of the heart by the Dublin committee of the medical section of the Fifth Meeting of the British Association for the Advancement of Science 111 in Dublin in 1932. London John Murray 1936.
9. ANDERSON D. Latest heart stenosis. A review. *Excerpta Medica* 21: 50 1951.
10. ALEXANDER M. J. L. AND THOMAS J. H. A. Aortic stenosis: the relation of all aortic stenosis to the aortic arch. Report of a case featured by primary atherosclerosis and of aortic atherosclerosis. *Am Heart J* 22: 833 1941.
11. ALLEN M. H. D. SHAW J. W. AND SWAN H. J. C. Ventricular septal defect in infancy and childhood. A clinical and physiological study of 13 cases. *Brit Heart J* 20: 548 1958.
12. ALLEN J. C. The wide patent ductus arteriosus. *Clinical Medicine* 35: 15 1935.
13. ALLEN M. M. RAPPAPORT M. B. AND
14. ALLEN M. M. RAPPAPORT M. B. AND
15. ALLEN M. M. RAPPAPORT M. B. AND
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phonocardiographic mensuration. Particularly is this true since the rapid deflections of higher frequency composition (above 100 cps) are, as a rule, used for making temporal measurements.

### Storage of Heart Sound Information

If one is to record on magnetic tape for subsequent analysis both oscillographically and spectrographically, it is desirable to have only as much low frequency attenuation as is necessary in order for the tape to accommodate the wide dynamic (i.e., intensity) range involved. Thereafter, the little altered signal can be put into the spectrograph (which can accommodate a wide intensity range) and the tapes can be used for teaching purposes; the necessary attenuation for low frequency and high frequency (Isthmian), stethoscopic and logarithmic (Rippert), oscillograms can be imposed between the tape and the oscillograph.

### Artifacts of Overloading

A square wave imposed on the spectrograph, as displayed as indicated in Figure 493. A transient with frequency content over the entire range encompassed by the instrument occurs both with onset (A) and offset (B) of the wave. If the square wave is of short duration an apparently unitary transient results. If a low frequency vibration is recorded on the tape at excessive gain there may be a cut off of its top so that in effect something closely approaching a square wave is produced. The spectrogram might be interpreted as representing a split sound. This appears to be what happened when the sounds of the Hufnagel valve were recorded (1077). The initial recording on tape was made in the operating room soon after placement of the valve in the aorta and with the microphone held by the surgeon close to the prothesis. The spectrogram shows splitting of both the sound of opening and that of closing. The

conclusion was arrived at earlier (1077) that there is asynchronous impact of the ball on the corners which hold the valve open in cardiac systole and that in closing the ball impinges first on one side of its circular seat and then on the rest. It is almost certain that the tape was "overloaded" in the initial recording and that artifactual splitting resulted by the mechanism described above. No such splitting is observed when the Hufnagel sounds are recorded from the surface of the chest without overloading. We have also observed artifactual splitting on this same bias when the valve closure sounds are recorded at very high gain. Figure 494 presents such a case. Both the tricuspid closure sound and the mitral closure sound appear to be slightly split. In this case the sounds were re-recorded at high gain in order to overcome the fore-shortening of the frequency scale which was an inevitable accompaniment of spreading the time scale by the method then available.

Several times earlier (p 160 footnote p 501) reference has been made to the risks of introducing artifactual high frequency components when the phonocardiographic system is overloaded by intense low frequency components at any stage from the microphone to the final display. Junggren (959) points out that this can happen with filter systems such as used in the Münchheimer Stordal method. Rodbard (1293) uses a sharp low cut filter to eliminate components below 1000 cps. He then displays high frequency 'spikes' which he interprets as evidence that cardiac vascular sounds (both heart sounds and murmurs) are basically a series of closely spaced transient. Although an extensive critique of the method and results is not possible one would suspect that the 'spikes' are high frequency artifacts generated by 'hitting' the microphone or the filter excessively hard with low frequency components.



- 27 ARAYANIS C AND IUSADA A A Obstructive and relative aortic stenosis Differential diagnosis by phonocardiography *Am Heart J* 51: 32 1957
- 28 ARMSTRONG I I ADAMS W I TRACERMAN J J AND TOWNSEND I W The Cruveilhier-Baumgarten syndrome Review of the literature and report of two additional cases *Ann Int Med* 16: 113 1942
- 28A ARINCIN N I AND ZENKEVICH T S Half a century in the application and further development of Korotkov's auditory method of determining blood pressure *Sechenov Physiol J USSR* 43: 81 1957
- 29 ARVIDSSON H KARNELL J AND MÖLLER T Multiple stenosis of the pulmonary arteries associated with pulmonary hypertension diagnosed by selective angiocardiography *Acta radiol* 44: 209 1955
- 30 ASH R I Evolution of rheumatic disease in childhood *Pennsylvania M J* 44: 484 1941
- 31 ASH R AND MURPHY L High ventricular septal defect and slight dextroposition of the aorta (Eisenmenger complex) associated with deformed aortic valve simulating patent ductus arteriosus *J Pediatr* 37: 249 1950
- 32 ASHMAN R Normal duration of the Q-T interval *Proc Soc Exper Biol & Med* 40: 150 1939
- 33 ASHMAN R AND HILL I *Essentials of Electrocardiography* The Macmillan Co New York 1937
- 34 ASHWORTH C T Atherosclerotic valvular disease of the heart *Arch Path* 42: 285 1946
- 35 ASKLEY J M Spontaneous rupture of a papillary muscle of the heart *Am J Med* 9: 523 1950
- 36 AURINGER W Über die Lautstärke des 1 Herz tones bei Arrhythmien *Ztschr Kreislaufforsch* 44: 195 1955
- 37 AURINGER W Über die veränderliche Lautstärke des Vorhof tones bei Galopprrhythmus *Wien Ztschr inn Med* 36: 163 1954
- 38 AZEVEDO A DE C BARRETTO NETTO M GARCIA A AND DE CARVALHO A A Patent ductus arteriosus and congenital mitral stenosis *Am Heart J* 45: 295 1953
- 39 AZEVEDO A DE C ROUBACH R VERA TOLEDO A AND DE CARVALHO A Diagnosis and surgical treatment of congenital aortic septal defects *Acta cardiol* 9: 1 1954
- 40 BAFFES T G JOHNSON F R POTTS W J AND GIBSON S Anatomic variations in the tetralogy of Fallot *Am Heart J* 45: 657 1953
- 41 BACGENSTOSS A H AND ROSENBERG I F Unusual cardiac lesions associated with chronic multiple rheumatoid arthritis *Arch Path* 37: 54 1944
- 42 BAINSON H T AND NEUMAN E V Diagnosis and surgical removal of intracavitary myxoma of the right atrium *Bull Johns Hopkins Hosp* 93: 150 1953
- 43 BAILEY C P AND BOLTON H I Results of mitral commissurotomy *N Y J Med* 56: 525 1956
- 44 BAILEY O T AND HICKAM J B Rupture of mitral chordae tendineae Clinical and pathologic observations on severe cases in which there was no bacterial endocarditis *Am Heart J* 28: 518 1944
- 44A BAIN R C EDWARDS J I SCHIFFRICH C H AND CERACI J I Right sided bacterial endocarditis and endarteritis A clinical and pathologic study *Am J Med* 24: 93 1958
- 45 BAKER C BRISTON W D AND CHANNELL G D Infectious disease *Guy Hosp Rep* 99: 24 1950
- 46 BAKER I A SIRACE H B AND WHITE P D The clinical significance of loud aortic and aortic systolic heart murmurs without diastolic murmurs *Am J M Sc* 206: 31 1943
- 47 BALL J D DAVIES J N P AND WILLIAMS A W Indomycardial fibrosis *Lancet* 1: 1049 1954
- 48 BALLANTYNE J W *Manual of Internal Pathology and Hygiene* Vol 2 p 509 Green Edinburgh 1902
- 49 BAYKS J T Perforation of the aortic valve—loud musical murmur *Dublin Hosp Gazette* 14: 33 1957
- 50 BARNER H AND OSBORN C R A case of mitral stenosis the result of trauma *Guy Hosp Rep* 87: 510 1937
- 51 BARBER J M MACDONALD O AND WOOD I Atrial septal defect with special reference to the electrocardiogram the pulmonary artery pressure and the second heart sound *Brit Heart J* 10: 277 1950
- 52 BARKER A C ROL B B AND RICHARDSON C S Relation of valvular lesions and of exercise to aortic pressure work tolerance and to development of chronic congestive failure in dogs *Am J Physiol* 169: 384 1954
- 53 BARRÉ I Le bruit de galop *Semaine méd* 13: 473 1893
- 54 BARRÉ I Le souffle cardio pulmonaire dia-liquide *Bull et mém Soc méd hip Paris* p 295 1896
- 55 BARKER I F Electrocardiography and phonocardiography *Bull Johns Hopkins* p 21 308 1910
- 56 BARKER P S AND JOHNSTON F D Chronic pericarditis with effusion *Circulation* 2: 134 1950
- 57 BARKER P S JOHNSTON I D AND WILSON F A Duration of systole in hypocalcemia *Am Heart J* 14: 82 1937
- 58 BARLOW J AND SHILLINGFORD J Amyl nitrite in the differentiation of regurgitant and ejection systolic murmurs *Abstract Brit Heart J* 10: 585 1957 *ibid* 20: 162 1958
- 59 BARRITT D W Simple pulmonary stenosis *Brit Heart J* 16: 351 1954

- 191 BURT W AND FRY F A Prognostic value of life insurance mortality investigation J A M A 169 36 1942
- 192 BURT W AND CALO A Anetres me cironle du front particuliere phonocardiographique de restitue Arch mal coeur 32 308 1939
- 193 BURT W AND ORFENHEIMER E T (Alloprhythm in heart nation Arch Int Med 11 166 1944
- 194 BURT W Die Entdeckung der Herzgeräusche. Ergebnisse und Mel u Kinderh 30 168 1938
- 195 BURT W Herzhinterwand und oesophageale Auskultation des Herzens (Abhandlungen aus dem Cemitat der Medizin 1937
- 196 BURT W Zur Entdeckung der muskularischen Herzgeräusche Wien Arch f inn Med 28 13 1939
- 197 BOYMAN CISTER R J AND WATKINS C H M Continuous murmurs without present diastolic apertures Lancet 2 779 1930
- 198 BRYNE J A AND LEVINE S A The prognosis in potential rheumatic heart disease and rheumatic mitral insufficiency Am J M Sc 39 64 34
- 199 BOOTHBY W M AND REYNOLDS D H Increase in circulation rate produced by exophthalmic goiter Arch Int Med 53 547 1930
- 200 BOUTARD J R AND ROUEDEL F M Intra thoracic auscultation in pneumothorax hyper Mel A Surg 4 34 1946
- 201 BOWLEY J M CHAMBERLAIN L A H HAMILTON W F KERR W J AND WINTER C J Recommendation for human blood pressure determination by phigmonanometer Circulation 4 303 1951
- 202 BRYAN C V H DAKES E D MOTT J C AND REYNOLDS B R The contraction of the ductus arteriosus early oxygen addition a physical neonatal limit J Physiol 132 301 1946
- 203 BERTER W AND MILLER I Effect of electro phonocardiogram in Arbeit network Cardiolgia 27 134 1942
- 204 BRILLAUD J H Traité clinique des Maladies du Coeur Paris 1938
- 205 BRILLAUD J B 7 ansé linique des maladies du coeur Vol I (a) p 393 (c) p 18 (d) p 264 (e) p 101 (f) p 13 Paris 1940
- 206 BRYNE C Cardiac signs in young adults with special reference to functional murmurs Lancet 251 1 1946
- 207 BRYNE C Clinical signs of functional heart disease Minnesota Med 29 617 1934
- 208 BRYNE C Some heart murmurs made Lancet 1 1087 1936
- 209 BOWDITCH V A L Jeanette's ponder of Hensen Jager all B with a Vol I p 30 Houghton Mifflin (C) Boston 1909
- 210 BOWER B D (FARR M J W D BROWN A L AND RAY C C Two cases of congenital mitral stenosis treated by valvotomy Arch Dis Childhood 28 91 1953
- 211 BRYER A H LAKES R W AND WINTER C J The characteristics of normal heart sound recorded by direct method Am Heart J 29 157 1944
- 212 BRYER A H AND CHAMBERLAIN W L Clinical observation
- 213 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 214 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 215 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 216 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 217 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
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- 219 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
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- 281 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 282 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 283 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 284 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 285 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 286 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 287 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 288 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 289 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 290 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 291 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 292 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 293 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 294 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 295 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 296 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 297 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 298 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 299 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944
- 300 BRYER A H AND CHAMBERLAIN W L Clinical observation in the diagnosis of pulmonary hypertension Am Heart J 63 461 1944



- diagnosis of congenital anomaly of the heart. Case report with a review of the literature. *Pediatrics* 3: 504 1949
- 97 BERRY M R *Vascular Clinics* VII The mechanism and prevention of impairment of aortic sounds during determination of blood pressure of standing patients. *Proc Staff Meet Mayo Clin* 15: 699 1940
  - 98 BERTHIER AND DE LONT REAULT Souffle diastolique intense irradiations ou eucardioinsuffisance aortique. *Bull et méém Soc méd hôp Paris* 1: 274 1934
  - 99 BERTIN R J *Traité des Maladies du Cœur et des gros Vaisseaux* pp 176-177 Jun 1924
  - 100 BERTRAND C A MUIR I G AND HORVICK R A study of heart sounds and murmurs by direct heart recording. *Circulation* 13: 49 1956
  - 101 BERTERMAN I M M Phonocardiography in acute rheumatic carditis. *Brit Heart J* 17: 360 1955
  - 102 BERTERMAN I M M The use of phenylephrine to aid in cultivation of early rheumatic diastolic murmurs. *Brit Med J* 2: 205 1951
  - 103 BIANCHI A *The Phonendoscope and its Practical Applications* Translated by A C Baker Philadelphia 1938
  - 104 BICHFI C AND MOZER J J Infarctus perforant de la cloison interventriculaire. *Bull et méém Soc méd hôp Paris* 61: 1564 1935
  - 105 BIEHL J I AND SIMON D I Auricular sounds in a case of auricular flutter. *Am J Med* 13: 134 1953
  - 106 BIERRE W I BONE H C AND LOCKHART M L Use of the electro kethograph for recording heart sound. *J A M A* 104: 628 1935
  - 107 BILLING A On the auscultation and treatment of affections of the heart. *Imcet* 2: 194 1832
  - 108 BILLING ARCHIBALD (Obituary with portrait). *Med Cire London* 1: 243 1842
  - 109 BIRBAK C AND CRAPOORD C Arteriovenous aneurysm on the pulmonary artery simulating patent ductus arteriosus. *Botalli Thorax* 2: 65 1947
  - 110 BISHOP I AND LOCIE B Internal rupture of heart causing systolic murmur and thrill. *J A M A* 144: 757 1950
  - 111 BISHOP R C Delayed closure of the ductus arteriosus. *Am Heart J* 44: 639 1952
  - 112 BJÖRKMAN S I On the occurrence of a valvular murmur over greatly enlarged pleura. *Acta med Scandinav* 145: 79 1953
  - 113 BLACKNEY R B SIVCLAIR SMITH B C CALVER A J HOLLIDAY J H AND MADDOX J K Electrocardiogram a report of 5 cases. *Anthriscian Ann Med* 1: 26 1952
  - 114 BLACKFORD C M AND PARKER F P Pulmonary stenosis with bundle branch block. report of a case with sound tracings and emicardial studies of the conduction bundle. *Arch Int Med* 67: 1107 1941
  - 115 BLACKMAN S S JR Syphilis of the mitral valve and membranous interventricular septum of the heart. *Bull Johns Hopkins Hosp* 57: 111 1935
  - 116 BLAIR H A WOOD A M AND YOUNG A C The relation of the Q-T interval to the refractory period the diastolic interval the duration of contraction and the rate of heating in heart muscle. *Am J Physiol* 132: 157 1941
  - 117 BLAND L F Cibot case No 4221. *New England J Med* 255: 44 1956
  - 118 BLAND I F JONES T D AND WHITE P D Disappearance of the physical signs of rheumatic heart disease. *J A M A* 107: 569 1936
  - 119 BLAND I F AND WHEELER I O Severe aortic regurgitation in young people. A long term perspective with reference to prognosis and prophylaxis. *New England J Med* 255: 607 1956
  - 120 BLAND I F WHITE I D AND JONES T D Development of mitral stenosis in young people with discussion of frequent misinterpretation of mid diastolic murmur at the cardiac apex. *Am Heart J* 10: 995 1935
  - 121 BLOCK W J PARKER R L AND EDWARDS J I Myxoma of left atrium clinically simulating mitral stenosis. report of case and pathologic study. *Proc Staff Meet Mayo Clin* 27: 361 1952
  - 122 BLOOM H J G Venous hum in hepatic cirrhosis. *Brit Heart J* 11: 313 1949
  - 123 BLOUNT S G BAILEY O J AND GRASSING G The persistent ostium primum atrial septal defect. *Circulation* 13: 499 1956
  - 124 BLOUNT S C JR McCORD M C AND CLEGG I J Ebstein anomaly. *Circulation* 15: 710, 1957
  - 125 BLOUNT S G JR SWAN H GENONI C AND McCORD M C Atrial septal defect clinical and physiologic response to complete closure in 5 patients. *Circulation* 9: 801 1954
  - 126 BLUMBERG B AND RACAN C Natural history of rheumatoid polydermatitis. *Medicine* 35: 1 1956
  - 127 BLUMBERG C Pericarditis epistenoecardia. *J A M A* 107: 178 1936
  - 128 BLUMHART H L AND FRANKS A C Two mechanisms in the production of Duroziez's sign. Their diagnostic significance and clinical test for differentiating between them. *J A M A* 100: 173 1933
  - 129 BLUMHART H I CARROLL S I AND CHILMAN D R Studies on the velocity of blood flow. III The circulatory response to thyrotoxicosis. *J Clin Invest* 9: 69 1930
  - 130 BLUMHART H I AND WEISS B Studies on the velocity of blood flow. IV The velocity of blood flow and other aspects of the circulation in patients with primary and secondary anemia and in two patients with polycythemia vera. *J Clin Invest* 9: 69 1931
  - 131 BOAS I P Evolution of calcareous aortic stenosis. *Circulation* 8: 142 1953

- 1 and origin of heart sound Acta cardiol 4 171 1949
- 18 BURGER H C KOOPMAN I J AND OVEREEM A I T On the analysis and the origin of heart sound (III) Acta cardiol 5 1 1950
- 19 BURGER H C VAN BRUNSWIEN A C W AND DAVENBERG F J Theory and experiment on rheumatic model of tension Circulation Res 4 45 1956
- 20 BURKE F D KIRKLEN J W AND LOWMAN J I Site of obstruction in pulmonary blood flow in the tetralogy of Fallot an anatomic study Proc Staff Meet Mayo Clin 26 498 1951
- 21 BURN A Observations on some of the most frequent and important diseases etc (a) pp 157-180 (b) p 95 1907
- 22 BURTON C C Improvement in construction of apparatus for demonstration of turbulence J Appl Physiol 6 19 1951
- 23 BURNELL C B Boston Personal communication
- 24 BURNELL C C The placenta as a modified AV fistula considered in relation to the circulation Am J Med Sci 195 1 1938
- 25 BURNELL C C AND MERRILL J Heart Disease and Pregnancy Physiology and Management Little Brown and Co Boston 1954
- 26 BUTTERWORTH J M Personal communication
- 27 BUTTERWORTH J B CHAMBERS M R AND McGRATH R Cardiac illustration in ludwig's diagnosis of principles of medicine Stratton New York 1950
- 28 BYSTROM J AND SALVATI H J Rupture of aortic aneurysm into the pulmonary artery Am J Med Sci 19 19 1950
- 29 CIBOT R C A multiple electrical tachycoscope for teaching purposes J A M A 181 1223 1923
- 30 CIBOT R C Theory of Diagrams Ed 9 p 19 Wm Wood New York 1951
- 31 CIBOT R C AND DUDLEY H F Frequency characteristics of heart and lung output J A M A 181 1793 1925
- 32 CIBOT R C AND LUCKE F A On the occurrence of diastolic murmur without closure of the aortic or pulmonary valve Bull John Hopkins Hosp 14 115 1903
- 33 CARRERA C I AND MONTGOMERY J R Systolic and diastolic loading of the heart II Physiology and clinical data Am Heart J 43 661 1952
- 34 CARRERA C F AND MONTGOMERY J R Systolic and diastolic loading of the heart II Electrocardiographic data Am Heart J 43 661 1952
- 35 CARRUTHERS A R De modis acutis et chronicis II in XIV Edited and translated by I F Frohman p 630 University of Chicago Press Chicago 1950
- 36 CAFFE Obituary of Rouquet J de conat an mel 33 11 1946
- 37 CAIN E F AND WAKE F R Diaphragmatic flutter with symptoms J Am A 131 105 1946
- 38 CAIRO A A elettrocardiografia dei toni atriali in un caso di flutter auricolare Cardiologia 3 14 1950
- 39 CAIRO A Contribuzione alla lettura del miocardiogramma dei toni atriali e ventricoli Cardiologia 3 14 1950
- 40 CAIRO A Contribuzione alla lettura del fonocardiogramma dei toni extra cardiaci Rev med franc May-June 1950
- 41 CAIRO A I casi extra cardiaci diastolici a proporzioni di efficacia polmonare normale e diastolici Atti del VIII Congresso della Societa Italiana di Cardiologia 30 31 March 1951 Atti Soc Ital cardi 13 197 1951
- 42 CAIRO A La phase de reaction ventriculaire (la phase et le cinquieme) rust du pour (Arch) Cardiologia 11 1951
- 43 CAIRO A Le couffage technique mu (ra) de lauffiance aortique Arch mal coeur 40 137 1951
- 44 CAIRO A Les Bruits de coeur et les valvulopathies Mon et Cie Paris 1950
- 45 CAIRO A Sur une variete peu connue et essentiellement benigne le rythme a trois temps Le cinquieme a trois pleurs pericardique Tu me mel 33 31 1947
- 46 CAMBERG C I Self-sustaining tetrapole New York Med Times 4 140 1950
- 47 CAMBERG M Latent ducty arterium Some notes on prognosis and on pulmonary hypertension Brit Heart J 17 515 1950
- 48 CAMBERG M Differential diagnosis of Fallot's tetralogy and simple pulmonary stenosis with a reversed interatrial shunt Brit Heart J 20 195 1950
- 49 CAMBERG M AND HATLEY J H The course and prognosis of coarctation of the aorta Brit Heart J 18 45 1956
- 50 CAMBERG M AND BRUNN R C The result of valvulotomy for simple pulmonary stenosis Brit Heart J 17 229 1950
- 51 CAMBERG M AND HATLEY J H The disappearance of the continuous murmur of patent ducty arteria Ann Hosp Rep 101 32 1950
- 52 CAMBERG M AND BRUNN R C I Indocyanine blue defect common atrioventricular canal and ostium primum Brit Heart J 18 403 1957
- 53 CAMBERG M AND HATLEY J H AND COOKER W B Congenital disease of the pulmonary valve Circulation 15 37 1957
- 54 CAVE C AND KATZNER R Acute aortic valve regurgitation (Am Hosp Rep 105 107 1956
- 55 CANNELL D F Congenital aneurysm of the interventricular septum report of two cases Am J Path 6 411 1950
- 56 CANNON W B Auscultation of the rhythmic

- Hemodynamic vectorcardiographic and electrocardiographic observations *Circulation* **13** 866 1956
- 166 BRAUNWALD I FISHMAN A P AND COURNAUD A Time relationship of dynamic events in the cardiac chambers pulmonary artery and aorta in man *Circulation Res* **4** 100 1956
- 167 BRAUNWALD I AND MORROW A G Method for detection and estimation of magnitude of aortic regurgitation flow Abstract *Circulation* **16** 862 1957
- 168 BRAUNWALD L AND MORROW A C The sequence of ventricular contraction in bundle branch block Presented at meeting of Am Heart Assn Cincinnati Oct 25 1956 Abstract *Circulation* **14** 915 1956
- 169 BRAUNWALD I MOSCOWITZ H I AVRAM S S LASSER R P SALTIN S O HIMMELSTEIN A RAVITCH M M AND GORDON A J The hemodynamic of the left side of the heart as studied by simultaneous left atrial left ventricular and aortic pressures particular reference to mitral stenosis *Circulation* **12** 69 1955
- 170 BRAUNWALD I MOSCOWITZ H L AVRAM S S LASSER R P SALTIN S O HIMMELSTEIN A RAVITCH M M AND GORDON A J Timing of electrical and mechanical events of the left side of the human heart *J Appl Physiol* **8** 309 1955
- 171 BRAUNWALD L SARNOFF S J AND STAINSBY W N Determinants of duration and mean rate of ventricular ejection *Circulation Research* **6** 319 1958
- 171A BRAUNWALD I WELCH C H JR AND SARNOFF S J Hemodynamic effects of quantitatively varied experimentally mitral regurgitation *Circulation Research* **5** 539 1957
- 172 BRECHER C A *Venous Return Curve A Study* New York 1956
- 173 BRECHER C A AND HUBAY C A Pulmonary blood flow and venous return during spontaneous respiration *Circulation Res* **3** 210 1955
- 174 BRICHETEAU I Observation d'hydro pneumopéricarde accompagnée d'un bruit de fluctuation perceptible à l'oreille *Arch Gen de Méd* **11** 334 1844
- 175 BRIDGMAN F W Notes on a normal presystolic sound *Arch Int Med* **14** 475 1914
- 176 BRIDGMAN L W Observations on the third heart sound *Heart* **6** 41 1915
- 177 BRIGDEN W AND LEATHAN A Mitral incompetence *Brit Heart J* **15** 55 1953
- 178 BRISKIER A *Cardio charting Universal Method of Recording Heart Auscultation* Macmillan New York 1957
- 179 BROCK R C The surgical and pathological anatomy of the mitral valve *Brit Heart J* **14** 489 1952
- 179A BROCK SIR RUSSELL Functional obstruction of the left ventricle (acquired pulmonary stenosis) Guy & Ho p Rep **106** 221 1957
- 179B BROCK SIR RUSSELL *The Anatomy of Congenital Pulmonary Stenosis* New York Hoeber Harper, 1957
- 180 BROCKBANK I M The crescendo murmur of mitral stenosis *Brit M J* **2** 509 1959 and 15 9 1909
- 181 BROCKBANK I M *The Diagnosis and Treatment of Heart Disease Practical Points for Students and Practitioners* Paul B Hoeber New York 1919
- 182 BROCKBANK I M *The Diagnosis and Treatment of Heart Disease* 1d 4 1920
- 183 BROCKBANK I M *The Murmurs of Mitral Disease* Edinburgh & London 1899
- 184 BROFMAN B I AND FELI H The diagnosis of congenital subaortic stenosis *Circulation* **6** 817 1952
- 184A BROOKS H ST J Two cases of an abnormal coronary artery of the heart arising from the pulmonary artery with some remarks upon the effect of this anomaly in producing aortic dilation of the vessel *J Anat* **10** 26 1886
- 185 BROTSCHER L AND DELCHER D C The systemic blood flow in congenital heart disease with an examination of the validity of the cardiac index *Clin Sci* **15** 441 1956
- 186 BROWN J W *Congenital Heart Disease* 1d 1 Stiple Press London 1950
- 187 BROWN I The story of the tethoscope In *The Story of Clinical Pulmonary Tuberculosis Ch XIII* Baltimore Williams & Wilkin 1941
- 188 BURNER A J AND BIRCHELL H B Kinking of aortic arch (pseudocoarctation subclinical coarctation) *J A M A* **162** 1445 1956
- 189 BUTLER J F (a) Experiments to determine the origin of the respiratory sound *Proc Roy Soc London* **37** 441 1884 (b) On the breath sounds in health and disease *St Birth Ho p Rep* **21** 191 1885
- 190 BURNIN J J AND MCEWEN C Tophus of the mitral valve in gout *Arch Path* **29** 700 1940
- 191 BURR W H The sound produced by an embolus passing through the heart *J A M A* **97** 101 1931
- 191A BURCH G F 1 *Primer of Cardiology* 1st & Felinger Philadelphia 1953
- 192 BURCHELL H B Total anomalous pulmonary venous drainage clinical and physiologic pattern *Proc Staff Meet Mayo Clinic* **31** 161 1956
- 193 BURCHELL H B AND EDWARDS J I Aortic aneurysm with communications into right ventricle and associated ventricular septal defect *Proc Staff Meet Mayo Clinic* **26** 336 1951
- 194 BURCHELL H B AND EDWARDS J I Rheumatic mitral insufficiency *Circulation* **7** 747 1953
- 195 BURGER H C AND KOOPMAN I J On the anky-

- 54 COCHRAN R C AND KING A I Measurement of noise stature (Lancet) 233 379 1936
- 55 COCHRAN R C AND KING A I Verification of noise stature in terms of the primary standard J Int Elec Engng (of England) 81 57 1937
- 56 CLAIRBORNE T S AND HICKMAN W A Aortic pulmonary artery communication through the lungs: Report of a case Circulation 34 1946 1956
- 57 CLARK A Lectures on Diseases of the Heart Birmingham & Co New York 1881
- 58 CLARK H C Excision of Aortic Root mortem appearance Boston Med & Surg J 88 490 598
- 59 CLARK R J AND FERMINGER H Correlation of the aorta associated with stroke Adam syndrom complete heart block and bicuspid aortic valve New England J Med 240 110 1949
- 60 CLARK R J AND WHITT I D Congenital aortic defect of membranous portion of ventricular septum associated with heart block ventricular flutter Adam stroke syndrome and death Circulation 8 770 1957
- 61 CLARK J S KELLY J I AND BAUER W Rheumatic aortitis with aortic regurgitation Am J Med 22 540 1957
- 62 CLEVELAND W L CANNAN T B CLEVERLEY J F AND STEINER H F Correlation of the aorta Brit Med J 2 303 1956
- 63 CLIPPING Report Brit J Adv Sci 1950
- 64 CLERC A ASSEUR R AND SOULAS WILLIAMS le galop avec variation chronologique du bruit irradié l'apnée l'allongement subit par le passage au court l'un du bruit au bruit ventriculaire en ombre Arch mal coeur 33 193 1940
- 65 Clinical pathological inference Am J Med 34 820 1963
- 66 CUNNEN P W A study of the random ocular reaction to epinephrine Bull John Hopkins Hosp 33 268 1970
- 67 CREBENTZ B HARTLEY R M FERREN M I CLEVELAND A AND RICHARD D W Jr The relationship between electrical and mechanical event in the cardiac cycle of man Brit Heart J 12 1 1951
- 68 CROFT J H AND MILLER S Lieball Trust New England J Med 255 359 1956
- 69 CULIN A E The origin of the presystolic murmur Brit Med J 2 1153 1949
- 70 CULLEN R A AND CULLEN A B The aortic diastolic murmur in aortic insufficiency Bull John Hopkins Hosp 33 353 1949
- 71 COLLIER J De diversis methodis explorationis a) strum et de strum applicatione ad nosologiae morbi thesaurum translatus Forster and included in his English edition of the same pp 64 and 115 1874
- 72 COLMAN J Electrical alternans Case report and comments on the literature Am Heart J 55 417 1958
- 73 COMPTON J AND COLLETTI A Modification of phonocardiogramme and electrocardiogram pour l'étude mitrale Arch mal coeur 3 76 1951
- 74 CONNER I A Certain acoustic limitation of the stethoscope and their clinical importance Trans Am Phys Soc 22 117 1907
- 75 CONNOR J A Note on the occurrence of an unusual form of gall rhythm Am Heart J 2 314 1906-7
- 76 CONTRA A W Significance of systolic aortic murmurs in the elderly New England J Med 228 479 1953
- 77 CONTRA S Ventricular gallop in mitral stenosis its mechanism and significance Am Heart J 64 216 1957
- 78 CONTRA S AND LUCAS A A Modification of the heart sound in loud brachyochia J Mount Sinai Hosp 19 70 1954
- 79 CONTRA S MILLER R A AND SHANNON J Relative pulmonary stenosis Am Heart J 23 559 1957
- 80 CONTRA S MILLER R A WINTER R A AND LECHE W J Syndrome of tricuspid arteriosus communis with large pulmonary flow Resistance to patent ductus arteriosus Abstract Circulation 14 971 1957
- 81 COOPER D C McNAMARA D C AND LATHAM J R Aortic-pulmonary septal defect diagnosis and surgical treatment Surgery 42 101 1957
- 82 COOPER H S BARKS H T AND HANCOCK R Angiographically in congenital heart disease of cyanotic type with pulmonary stenosis or atresia I Ch effects on the tetralogy of Fallot and patent tricuspid arteriosus Radiology 32 379 1951
- 83 COOPER R S AND GROSS R D Had (off) of the Heart and Great Vessels The William and Williams Co Baltimore 1954
- 84 COOPER C F Brit Med J 23 107 1957
- 85 COOPER C F Pneumatic Heart Disease p 140 Wm Wood New York 1954
- 86 COOPER C W JR AND STRAIN H M A fetal heart rate meter Am J Obstet Gynec 73 190 1957
- 87 CORRISSAN D J Permanent patency of the mouth of the aorta or the junction of the aortic valves Edinburgh Med & Surg J 37 22 1937
- 88 CORRISSAN D J Inquiry into the cause of bruit de soufflet and first element systolic Lancet 2 133 1959
- 89 CORRISSAN J A F auscultation mitrale et les

- sounds produced by the stomach and intestine  
*Am J Physiol* **14** 339 1905
- 233 CALES R Malignant endocarditis lasting over six months without bruit *Brit M J* **2** 1925 1907
- 234 CARDI I Functional changes of the heart during hypothermia *Angiology* **7** 171 1956
- 235 CARLSON I I Gallop rhythm in children studied by means of calibrated phonocardiography. An experimental and clinical investigation *Acta paediat* **33** suppl VI 1946
- 236 CARLISLE H Observations on the motions and sounds of the heart. Report of Third Meeting (Cambridge) of Brit Ass Adv Sc 1934
- 237 CARON Tumeur polypiforme développée dans l'oreillette gauche et plongeant dans l'orifice auriculo-ventriculaire gauche: quelle résection? *Bull Soc anat de Paris* **28** 77 1954
- 238 CARROLL D Non traumatic aortic valve rupture *Bull Johns Hopkins Hosp* **89** 300 1951
- 239 CARSTENSEN I I, KAM I and SCHWAN H P Determination of the acoustic properties of blood and its constituents *J Acou Soc Am* **25** 296 1953
- 240 CARSWELL R Cited by Bouilland (140)
- 241 CARTER I Du son de percussion du thorax. *Arch de physiol norm et path* **7** 15 1895 also *New England J Med* **208** 355 1958
- 241A CARY F H and HUBBARD J W Acquired inter-ventricular septal defects secondary to trauma *Clin Res* **6** 126 1958 also *New England J Med* **258** 355 1958
- 241B CASE R B, MORROW A C, STAINSBY W and NESTOR J O Abnormal origin of the left coronary artery. The physiologic defect and suggested surgical treatment *Circulation* **17** 1062 1958
- 241C CASSEI W C, SHUTTELL J A JR, LILLY F H JR and BRIDGER A J Arteriovenous fistula of the splenic vein producing aortic Circulation **18** 1077 1957
- 242 CASSELLS D F Cardiovascular murmur in infant and children *Med clin N Am* **41** 75 1957
- 243 CASPARY M A Abdominal carcinomatosis with probable adrenal involvement *Proc Roy Soc Med* **24** 920 1931 **27** 220 1934
- 244 CASTENFORS H, PÖRFF I C and RUDENWALD, B The hydrodynamics of aortic valve stenosis: experiments with a special model *Cardiologia* **25** 37 1954
- 245 CASTEL M Les souffles méotiques. *Arch mal coeur* **26** 444 1933
- 246 CASTLEMAN H and TOWNE A W Case report of the Mauthner's General Hosp. Marked fenestration of aortic cusps leading to aortic regurgitation. *New England J Med* **245** 911 1951
- 247 CECIL R C, PARKER C I and PORTER W H Bacterial endocarditis—report of a case in which a true murmur did not appear and did appear *Am Heart J* **36** 934 1938
- 248 CERABINI G Der Mechanismus der halbmondförmigen Herzklappen. Leipzig the 18 18 Cited by Cilo (221) and by Henderson and Johnson (670)
- 249 CHAPMAN J S Spontaneous irruption of air from the lung pneumomediastinum *Am J Med* **18** 547 1955
- 250 CHATELAIN A Mémoire sur plusieurs cas remarquables de défaut de synchronisme des battements et des bruits de ventricules du coeur. *Arch gén de méd* **39** 393 1838
- 251 CHARTON A, MINOT C and BENOIST M Les bruits normaux du coeur du cheval. Étude phonocardiographique. *Bull Acad vet Fr* **18** 218 1943
- 252 CHAUVEAU A Mécanisme et théorie générale des murmures valvulaires ou bruits de souffles après l'expérimentation. *Compt rend Acad d e* **46** 539 1858
- 253 CHAUVEAU A and FAIVRE J Nouvelle recherche expérimentale sur les mouvements et les bruits du coeur. *Caz méd de Paris* 1856
- 254 CHIFFER S N and DIEHLADE F R Studies on the electrical systole (Q-T interval) of the heart. IV. The effect of digitalis on its duration in cardiac failure. *J Clin Invest* **11** 1241 1932
- 255 CHIFFER S N and ILLIC R C Studies in the electrical systole (Q-T interval) of the heart its duration in normal Chinese. *Chinese J Physiol* **4** 191 1930
- 256 CHENG T O, SUTTON C C and SUTTON D C Cruveilhier-Baumgarten syndrome. Review of the literature and report of a case. *Am J Med* **17** 143 1954
- 257 CHESTER W Patent ductus Botalli with subsequent bacterial endocarditis and recovery. *Am Heart J* **18** 492 1937
- 258 CHESTERMAN J T and WHITAKER W Surgical treatment of acquired tricuspid stenosis. *Thorax* **10** 321 1955
- 259 CHLAPAR H Ueber Netzbildungen im rechten Vorhofe des Herzens. *Beitr z path Anat u z allg. Path* **11** 1897
- 260 CHILLES N H, SMITH H I, CHRISTIANSON A and CERARI J F Spontaneous healing of subacute bacterial endocarditis with closure of patent ductus arteriosus. *Proc Staff Meet Mayo Clinic* **38** 520 1953
- 261 CHIN I I and ROSS D N Myxoma of the left atrium. Successful surgical removal under hypothermia. *Brit M J* **1** 1447 1967
- 262 CHISHOLM D R Trigonization of the mitral valves and its relation to certain types of systolic murmurs. *Am Heart J* **18** 361 1937
- 263 CHRISTIAN H A Discussion. *Tr A Am Phys Ther* **50** 284 1935

- atrial septal defect Acta med Scandinav 160  
177 1950
- 331 DAVID C P H MCCRACKEN B H AND Mc  
LIVEN D J S Congenital coronary arterio-  
venous aneurysm Brit Heart J 17 569 1955
- 332 DAVIES J A I AND BALL J D The pathology  
of endomyocardial fibrosis in Uganda Brit  
Heart J 17 33 1955
- 333 DAVIES J A I AND FISHER J A Correlation  
of the aorta double mitral A V orifice and leak-  
ing cerebral aneurysm Brit Heart J 5 197  
1951
- 334 DAVIES J C TROUT R C SUMNER J C  
AND CLIVER R I A simple mechanical pul-  
soduplicator for cinematography of cardiac valves  
in action Ann Surg 143 544 1956
- 335 DAVI C JR DILLON R F FELL E H AND  
CULL B M Anomalous coronary artery  
mimicking patent ductus arteriosus J A M A  
180 104 1956
- 336 DAVI F W JR AND ADRI I C Mitral  
stenosis in the infant New England J Med  
251 99 1954
- 337 DAVIS J AND MCGEE S D Preliminary re-  
port on an investigation of foetal electrocar-  
diography and foetal teichography M J  
Australia 41 501 1954
- 338 DAVIS C S MOTT J C AND WIDENBURG J  
G The cardiac murmur from the patent ductus  
arteriosus in newborn lambs J Physiol 228  
344 1950
- 339 DAVIS G S MOTT J C AND WIDENBURG J  
G The patency of the ductus arteriosus in  
newborn lambs and its physiological con-  
sequences J Physiol 228 361 1955
- 340 DAVIES P M An histological sketch of the val-  
vula experiment Bull Hist Med 24 790  
1949
- 341 DEAN A L JR The movement of the mitral  
cup in relation to the cardiac cycle Am J  
Physiol 100 900 1916
- 342 DEBACH C CALO A AND ALMANZA C Sur  
un cas sporadique de maladie de Friedreich  
avec cardiopathie et trouble du développe-  
ment osseux Arch mal coeur 28 579 1915
- 343 DE CATER A Mofalite musicale du souffle  
cardiaque Tre eméd 1 125 1940
- 344 DECHAMBRE A Dictionnaire encyclopédique des  
sciences médicales 5 (ser III) 33 157
- 345 DECHERD C M JR AND BEARD O W Func-  
tional mitral diastolic murmur Texas Rep  
Biol & Med 4 113 1946
- 346 DEITE G W LUTHERELL A WALLACE J D  
BROWN J R JR AND LEWIS D H Place of  
intracardiac phonocardiography in the diag-  
nosis of heart disease in man (abstract) Circu-  
lation 18 81 1957
- 347 DE SAUTELLE W T AND CREE F G The rela-  
tion of the papillary muscles to mitral regurgi-  
tant murmurs Arch Int Med 8 731 1911
- 348 DETTMER R A JR AND WIDENBURG J C  
H Factors in the etiology of congestive peri-  
carditis Circulation 12 30 1950
- 349 DETWEILER D H Auricular fibrillation in  
horses J Am Vet M A 126 4 1950
- 350 DEWITT D J AND MANNING J F A foetal  
phonocardiograph Electronic Engineering 29  
310 1950
- 351 DEXTER I Atrial septal defect Brit Heart J  
11 709 1950
- 352 DUFFY D A H AND VAN NIEUWSTADT C  
I C Diagnosis of congenital aortic septal  
defect The origin of two cases and special  
emphasis on a method which allows an accurate  
diagnosis by means of cardiac catheterization  
Circulation 13 58 1956
- 353 DICKENS J RABER C T AND CLIFFORD H  
Dynamic pulmonary regurgitation associated  
with a bicuspid valve Ann Int Med 48 451  
1958
- 354 DICKSON W H On occurrence of mitral  
mitral murmurs in connection with aortic  
stenosis Medicine Chir Tr 80 409 1897
- 355 DICICHELMO L AND CATTANEO M Rank-  
ing of the aorta report of two cases Acta  
radiol 44 193 1950
- 356 DIZIEL A I Two cases of congenital heart  
disease in which the diagnosis was made before  
birth Am J Obst & Gynec 27 170 1911
- 357 DICK W Further evidence of the purely val-  
vular origin of the first and third heart sound  
Am Heart J 30 33 1915
- 358 DICK W Heart Sound Cardiac Pulsation  
and Crises in the L of Kahan Press  
Lawrence 1957 (a) p 36 (b) p 44 (c) p 44
- 359 DICK W Loud presystolic sound over the jugu-  
lar veins associated with high venous pressure  
Am J Med 20 453 1956
- 360 DICK W Mode of production of the first heart  
sound Arch Int Med 51 73 1931
- 361 DICK W Personal communication
- 362 DICK W The role of increased hepatic arterial  
flow in the portal hypertension of cirrhosis  
Tr & Am Livermen 27 30 1912
- 363 DICK W CRANELL F AND TALKIN F The  
physiologic third heart sound its mechanism  
and relation to protodiastolic gallop Am  
Heart J 50 419 1955
- 364 DUNELAT J AND DALLAINE F Traité des  
cardiopathies congénitales Masson & Cie  
Paris 1954
- 365 DUNN F R Linn University Ca Personal  
communication
- 366 DUNN C T Editorial New horizons in cardio-  
vascular roentgenology Am J Roentgenol  
76 817 1956

- lions organiques du coeur et des gros vaisseaux p 236 Paris 1806
- 298 CORVISART J N L'œu sur les maladies et les lésions organiques du coeur et des gros vaisseaux p 396 Paris 1818
- 299 COSH J A Patent ductus arteriosus with pulmonary hypertension Brit Heart J 15 423 1953
- 300 COSSIO P AND BERCONSKY I El primer ruido cardiaco y el sopho presistolico en la e trechez mitral con fibrilacion Rev argent de cardiol 10 162 1913
- 301 COSSIO P BERCONSKY I AND DAMBROSI R G Auricular and ventricular pericardial frictions Am Heart J 24 223 1912
- 302 COSSIO P DAMBROSI R G AND WARRFORD THOMSON H F The first heart sound in auricular and ventricular extrasystoles Brit Heart J 9 275 1917
- 303 COSSIO P AND FONCI E G Auricular sound Am Heart J 11 723 1936
- 304 COSSIO P AND LASCALBA M Premier bruit du coeur et bruit auriculaire Arch mal coeur 29 139 1936
- 305 COSSIO P LASCALBA M AND FONCI E C Alteration of the heart sound Arch Int Med 58 912 1936
- 306 COLLISHED N AND LITTLE T R Mitral epital defect in the aged Brit M J 1 74 1957
- 307 COLTIER N A JR AND PAIFFENHEIMER J R Development of turbulence in flowing blood Am J Physiol 159 401 1949
- 308 COUNIHAN T MESSER A I RAPPAPORT M B AND SPRACUE H B The initial vibrations of the first heart sound Circulation 3 730 1951
- 309 COUNIHAN T B RAPPAPORT M B AND SPRACUE H B Physiologic and physical factor that govern the clinical appreciation of cardiac thrills Circulation 4 716 1951
- 310 COURNAND A MOTLEY H I HINDELSTEIN A DRESDALE I AND RICHARDS D W Jr Latent period between electrical and pressure pulse waves corresponding to right auricular systole Proc Soc Exper Biol & Med 63 149 1946
- 311 COWEN E D H The phonocardiography of heart murmur Part II Clinical result and discussion Brit Heart J 11 360 1949
- 312 COWEN E D H AND LARUM D H The phonocardiography of heart murmurs Part I Apparatus and technique Brit Heart J 11 356 1949
- 313 COWPER W Of oscillations or petrification in the coats of arteries particularly in the valves of the great artery Phil Trans Roy Soc B 215 1703-1712
- 314 COX N Cardiac murmurs in infancy Brit M J 1 148 1914
- 315 CRADDOCK W I Diaphragmatic flutter with symptom resembling inguin pectoris J A M A 146 1315 1951
- 316 CRAWFORD C MAXIMILIAN E AND WIKLUND T Diagnosis and treatment of patent ductus arteriosus (Botalli) in connection with operated cases Acta chir Scandinav III 94 1914
- 317 CRAIG I Phonocardiographic studies in mitral stenosis New England J Med 257 630 1957
- 317A CROVASSE L L AND LOCHE R B The continuous murmur over the carotid bulb A new sign in the early recognition of carotid artery insufficiency Clin Res 6 125 1958
- 318 CROVELLIER J Note sur les mouvements et les bruits du coeur Gaz med 9 497 1841
- 319 CURREN J H BROWNELL C I AND ARONOW S An automatic blood pressure recording machine New England J Med 256 750 1957
- 320 CURREN J H THOMPSON W B RAPPAPORT M B AND SIRACE H H Clinical and phonocardiographic observations on the first murmur New England J Med 248 583 1953
- 321 CURTIS J A ETERSCHOFF R G AND BENNETT I I JR Acquired arteriovenous fistula complicated by Pseudomonas aeruginosa endocarditis and endocarditis Bull Johns Hopkins Hosp 101 140 1957
- 321A CUSHING H AND BAILEY P Tumors arising from the Blood Vessels of the Brain London Bullière Tindall & Cox 1928
- 322 DACOSTA J C Physical Diagnosis p 344 Saunders Philadelphia 1908
- 323 DADD J S AND HOYLE C Congenital aortic defect Brit Heart J 11 390 1949
- 324 DALEY R AND FRANKS R Massive dilatation of left auricle Quart J Med 18 91 1949
- 325 DALEY R McVILLIAN I K R AND CORRIE R Mitral incompetence in experimental auricular fibrillation J med 2 15 1955
- 326 DAISSARD NIELSEN T Studies on intracardiac auricular sounds Acta psychiat et neurol 14 69 1939
- 327 DAMMANN J F BERTHOUD M AND BING R J Patent ductus A pre entation of the syndrome of patency of the ductus arteriosus with pulmonary hypertension and a hunting of blood flow from pulmonary artery to aorta Bull Johns Hopkins Hosp 92 125 1953
- 328 DAMMANN J F AND SKIL C Patent ductus arteriosus in the absence of a continuous murmur Circulation 6 110 1952
- 329 DAVARAJ T J Ventricular epital defect simulating patent ductus arteriosus Brit Heart J 11 279 1956
- 330 DARENBURG C V Historia des sciences médicales Vol 2 p 614 J B Bullière Paris 1840
- 330A DAVIDSEN H C Pulmonary hypertension and incompetence with holodystolic murmur in

- figuration and amplitude. *Circulation* 8: 709-713, 1953.
- 401 EDDLEMAN L E JR, WILLIS R, REYNE T J AND HARRISON T R. The kymocardiogram. I. Method of recording precordial movement. *Circulation* 8: 370, 1953.
- 402 EDDLEMAN F F JR, WILLIS R, WALKER P P, CHRISTIAN C L AND PIERCE J R. Relation ship of the physiologic third heart sound to the jugular venous pulse. *Am J Med* 17: 15, 1954.
- 403 Editorial. Efficiency of the stethoscope. *JAMA* 148: 256, 1952.
- 404 Editorial. *London Med* 142: 1871.
- 405 EDWARDS J A AND LEVINE H D. Auscultation in the diagnosis of compression of the subclavian artery. *New England J Med* 247: 79, 1952.
- 406 EDWARDS J A AND LEVINE H D. The murmur of peripheral arteriovenous fistula. *New England J Med* 247: 40, 1952.
- 407 EDWARDS J A AND LEVINE H D. Peripheral valvular murmurs: Mechanism of production and diagnostic significance. *AMA Arch Int Med* 80: 284, 1952.
- 408 EDWARDS J F. Differential diagnosis of mitral stenosis: a clinical-pathologic review of simulating condition. *Lab Invest* 1: 3, 1953.
- 409 EDWARDS J F. Pathologic consideration in correlation of the aortic flow. *Staff Meet Mayo Clin* 22: 31, 1945.
- 410 EDWARDS J E. Pathologic features of Ebstein's malformation of the tricuspid valve. *Proc Staff Meet Mayo Clin* 28: 59, 1953.
- 410A EDWARDS J F. Anomalous coronary arteries with special reference to arteriovenous-like communication. *Circulation* 11: 1001, 1953.
- 411 EDWARDS J E AND BIRNELL H B. The pathological anatomy of leishmaniasis between the aortic root and the heart including aortic aneurysm. *Thorax* 12: 125, 1952.
- 412 EDWARDS J F AND BIRNELL H B. Second symposium on cardiovascular sound. *Circulation* in press.
- 413 EDWARDS J F, BIRNELL H B AND CHRISTENSEN A. Specimen exhibiting the essential lesion in aneurysm of the coronary artery. *Proc Staff Meet Mayo Clin* 31: 407, 1954.
- 414 EDWARDS J W AND SIMPSON T. Observations on pericardial thickening. *Tulsa Med* 46: 1939.
- 415 FINTHVEN W. Die Registrierung der menschlichen Herzne mittel der Gartengalvanometer. *Pflüger Arch* 111: 461, 1907.
- 416 FINTHVEN W. Ein dritter Herzton. *Pflügers Arch Geophysiol* 120: 31, 1907.
- 417 FINTHVEN W. Westeres über das Elektrokardiogramm. *183 rs Arch geophysiol* 122: 51, 1908.
- 418 FINTHVEN W AND CELIK M A J. Die Registrierung der Herzne. *Pflügers Arch geophysiol* 57: 61, 1911.
- 419 FINTHVEN H. Stethogram in cardiovascular syphilis as aid for early diagnosis. *Am J Syph* 36: 30, 1952.
- 420 FLODRUP F L, HULTCHEN H N AND WILMOR M F. The physiologic closure of the ductus arteriosus in newborn infant. *J Clin Invest* 34: 957, 1952.
- 421 FLODRUP F L AND HULTCHEN H N. Pulmonary stenosis with increased pulmonary blood flow. *Am Heart J* 43: 338, 1952.
- 422 FLODRUP F, SELZER A AND HULTCHEN H. Stenosis of a branch of the pulmonary artery: an additional cause of continuous murmur over the chest. *Circulation* 15: 571, 1952.
- 423 FLEISCHER C F, COLLIER F C, BOWEN B R AND CHAMBERLAIN W I. Electrokinographic studies of a tachycardia of ejection from the ventricle. *Am Heart J* 35: 971, 1948.
- 424 FLEISCHER R AND FALKENBERG J M. The heart in anemia. *New England J Med* 220: 913, 1939.
- 425 FLEISCHER R C, BROWN W J JR, HALLER I I JR AND HAMILTON W F. Physiology of excitation in experimental pulmonary regurgitation. *J Thoracic Surg* 30: 633, 1952.
- 426 FLEMING C I. The effect of pressure on the stethoscope on the intrathoracic sound. *Bull John Hopkin Hosp* 19: 49, 1908.
- 427 FLEISCHER R, HELL I C W AND LOWE K C. Unilateral membranous pulmonary venous occlusion pulmonary hypertension and patent ductus arteriosus. *Brit Heart J* 17: 71, 1955.
- 427A FLEISCHER R K, CALDWELL D AND BEHRER M R. Blood pressure studies in infants and children with an electric microphone. *J Clin Invest* 32: 1, 1953.
- 427B FLEISCHER D I. Rupture of aortic aneurysm into superior vena cava. *Arch Int Med* 88: 89, 1953.
- 428 FLEISCHER M A, HILLMAN C R, CALDWELL H P AND CLARK E. Regurgitation after open valvotomy of subaortic stenosis accompanying severe valvular pulmonary stenosis. *Am Heart J* 46: 876, 1953.
- 429 ENGLE M A, PIRKE T I B, BRINK C AND TALLID H B. The anatomy of the tricuspid valve. Report of three cases and analysis of the clinical syndrome. *Circulation* 1: 1946, 1950.
- 430 ENGLE M A AND TALLID H B. Valvular pulmonary stenosis with intact ventricular septum and patent foramen ovale. Report of illustrative case and analysis of clinical syndrome. *Circulation* 2: 481, 1951.
- 431 EPSTEIN N. The heart in normal infant and children. Incidence of precordial systolic murmurs.



- 365 DOTTER C T Second symposium on cardiovascular sound Circulation Res 3 51 1955
- 366 DOUBLE I J *Sémiologie Générale* Vol II p 31 Paris 1817
- 367 DOUGLAS D M Mitral regurgitation with mobile valve cusps Brit M J 1 191 1957
- 368 DOUGLAS J An extraordinary dilatation or enlargement of the left ventricle of the heart Tr Roy Soc London p 181 1715 (#345)
- 369 DOW J W AND DEXTER I J Circulatory dynamics in atrial septal defect Abstract J Clin Invest 29 809 1950
- 370 DOW J W LEVINE H D ELKIN M HAYNES I W HELIENS H K WHITTENBERGER J W FERRIS B G COODATE W T HARVEY W P LEINER I C AND DEXTER I J Studies of congenital heart disease IV Uncomplicated pulmonary stenosis Circulation 1 207 1950
- 371 DRESSER W Cardiac diagnosis without laboratory and palpation and percussion signs M Clin North America 34 721 1950
- 372 DRESSER W Pulsations of the wall of the chest V Pulsations associated with mitral regurgitation and aneurysmal dilatation of the left auricle Arch Int Med 60 663 1937
- 373 DRESSLER W AND KLEINER M Tie of the respiratory muscles Report of three cases and review of the literature Am J Med 18 61 1954
- 374 DUBILIER W JR TAYLOR T I AND STEINBERG I Aortic aneurysm associated with coarctation of the aorta Am J Roentgenol 73 10 1955
- 375 DUBOCZYK B O Loud millwheel murmur presumably caused by air embolism in a patient with pneumoperitoneum Am Rev Tuberc 70 1092 1954
- 376 DUCHOSAT P Nouvelles recherches graphiques sur le bruit de galop Arch mal coeur 28 345 1935
- 377 DUCHOSAT P Study of gallop rhythm by combination of phonocardiographic and electrocardiographic methods Am Heart J 7 613 1932
- 378 DUCHOSAT P AND BOLDREAU J I Clat acci dental du premier bruit du coeur dans la m eciation auriculo ventriculaire Arch mal coeur 27 232 1934
- 379 DUCHOSAT I W FERRERO C LEFEBVRE A AND URDANETA L Advance in the clinical evaluation of aortic stenosis by aortic pulsed record ing of the neck Am Heart J 51 861 1956
- 380 DUKES H H *The Physiology of Domestic Animals* Comstock Ithaca 1939
- 381 DUNN F J Interpretation of functional versus organic murmurs by cardiodynamic methods Nebraska M J 38 296 1953
- 382 DUNN F L AND DICKERSON W J Third heart sound possible role of pericardium in its production Circulation Res 3 51 1955
- 383 DUNN F I AND RAHM W I JR Electro stethography II New Method for study of precordial transmission of cardiodynamics Am Heart J 44 95 1952
- 384 DUNN F I AND RAHM W I JR Electro stethography III Crystal microphone characteristic at low frequencies for the study of cardiodynamics Am Heart J 45 519 1953
- 385 DUNN F I AND RAHM W I JR The piston phone Arch Otolaryng 60 301 1953
- 386 DUNN F I AND RAHM W I JR The problem of calibration in heart sound recording Am Heart J 46 237 1953
- 387 DUNN F I AND RAHM W I JR The visual study of heart vibrations and sounds as transmitted through the precordium J Insurance Med 4 34 1949
- 388 DUNN F I RAHM W E JR AND COCHRAN R M Electro stethography I Cathode ray visualization of lung chest sound Ann Int Med 16 521 1942
- 389 DUNN H C Congenital heart block Proc Roy Soc Med 45 456 1952
- 390 DUTRECHT D J Clinical observations on intestinal motility South African M J 28 91 1961
- 391 DURANT T M LONG J AND OHLHEIMER M J Pulmonary (venous) air embolism Am Heart J 33 209 1947
- 392 DIROZIEZ P I L'analyse de la maladie du coeur Du rythme pathognomonique du rétrécissement mitral Arch gén de méd 80 391 1869
- 393 DIROZIEZ P I Du double souffle intermittent aural comme signe de l'insuffisance aortique Arch gén de méd 17 417 and 588 1861
- 394 DIROZIEZ P I *Traité clinique des Maladies du Coeur* Paris 1861
- 395 DIROZIEZ P I *Arch Hôp* p 310 Paris 1869
- 396 DUSHANE J W Total anomalous pulmonary venous connection Clinical aspects Proc Staff Meet Mayo Clinic 31 171 1956
- 397 EASTMAN N J *Williams Obstetrics* Ed II p 260 Appleton Century Crofts Inc New York 1956
- 398 EASTER W Über einen sehr seltenen Fall von Ineffizienz der Valvula tricuspidalis bedingt durch eine angeborene hochgradige Mi schbildung der elbe Arch f Anat u Physiol p 335 1866
- 399 EASTMAN R W Sounds due to muscular contraction and their importance in cultivation of qualities of the first heart sound Am J Physiol 118 359 1937
- 400 EDDLEMAN I L JR WILLIS K CHRISTIANSON I FIERCE J R AND WALKER R J The kymocardiogram II The normal con

- ican edition) p 15 James Wel ter Philadelphia 1923
- 40 FORT A B HELLERSTEIN H K WOOD C AND KUTL H B Icteric congenital bicuspid pulmonary valve Clinical and pathologic study *Am J Med Sci* 20 4 195
- 41 FORTER H W Clinical lecture on rupture of the aortic valve from aneurysm *Med Times and Gaz* 2 65-656 656-656 1873
- 42 FOSTER H W On two cases of injury of the aortic valve from muscular exertion *Medical Press and Circ* Dec 19 1948 (2)
- 43 FOSTER J H AND SMITH I I JR The differential heart sound meter *Indiana Med J* 38 297 1944
- 44 FULTON J H SMITH I I JR AND FLEMING A J Cardiac vibrational intensity and cardiac output *Am Heart J* 35 93 1948
- 45 FULTON J H SMITH I I JR AND FLEMING A J Changes in cardiac vibrational intensity in response to physiologic stress *Am Heart J* 35 194
- 46 FOWLER N O AND FULTON H B JR Left ventricular of the infarcted interventricular septum Report of two cases one diagnosed by antemortem *Am J Med Sci* 215 271 1948
- 47 FOWLER N O MANNIX F I AND SCHEIDT W S Effect of partial pulmonary valvectomy *Circulation Res* 4 5 1956
- 48 FOXON C F H CRIPPS J AND LIND M Circulation in *Ischaemia* *Nature* 172 319 1953
- 49 FOXON C F H Problem of the blood circulation in vertebrates *Brit Rev* 30 136 195
- 50 FRIENTZ G Ueber die peristaltische Bewegung *Arch klin Med* 3 41 1905
- 51 FRANCH R H AND FRIEDMAN N O Ventricular septal defect related to myocardial injury of the heart Report of a case with histologic and ultrastructural studies *Am Heart J* 35 135 1948
- 52 FRANK O Die unmittelbare Registrierung der Herzneigung *Monatshchr Med Naturhchr* 61 273 1904
- 53 FRANKLIN R B AND FLECK B I The acute aortic aneurysm A review and classification of three cases *Medicine* 31 195
- 54 FRIEDRICH H A AND FRIEDMAN N O The stethophone an electrical telephone The Bell System Technical J 3 3 4 1944
- 55 FRIEDMAN A R AND LEVINE S A The clinical significance of the systolic murmur A study of 1000 consecutive non aortic cases *Ann Int Med* 6 13 1933
- 56 FRIEDMAN N O Rupture of aortic valve (symptomatic) *Brit Med J* 1 736 1879
- 57 FRIEDMAN A S Entail in auscultation of the heart *Mod Concept of Cardiovascular* 24 303 1955
- 58 FRIEDMAN B D MY W M AND SHEPHERD R S Orthostatic factors in jugular alternation *Circulation* 8 861 1953
- 59 FRIEDMAN B AND HATHAWAY B M Entailment of the semilunar cusps and functional aortic and pulmonary valve in ulcerative *Am J Med Sci* 24 519 1955
- 60 FRIEDMAN S AND HARRIS T N Aortic dilatation found in active rheumatic fever *J Pediatr* 3 633 1919
- 61 FRIEDMAN S ROME W A HARRIS T N Occurrence of innocent physiological cardiac sound in childhood *Pediatrics* 4 282 1913
- 62 FRIEDMAN ROME W A HARRIS T N Personal communication
- 63 FRIEDMAN R COLEMAN B CHART J VINCOW C FOSTER M AND CALLAGHAN I Lesions cardiaques à trois temps Phonocardiographie *J Med Sci* 1954 179 March 5 1955
- 64 FRIEDMAN R CALLAGHAN I AND BALESTIER C A pect variabilité du galop du 1<sup>er</sup> simple ou bivalent selon le degré de remplissage ventriculaire *Arch mal coeur* 2 12 1950
- 65 FRIEDMAN R CALLAGHAN I AND CHEN I Claquement et vibration de parietales contractives A propos d'un nouveau syndrome clinique *Arch mal coeur* 42 950 1949
- 66 FRIEDMAN R CALLAGHAN I AND FINAN Deux observations de faussement cardiaque métrique à trois temps avec frémiement *Arch mal coeur* 41 24 1948
- 67 FRIEDMAN R AND CHEN I Le syndrome du syndrome du syndrome *Arch mal coeur* 5 6 67 Mai Juin 1947
- 68 FRIEDMAN R CHEN I AND MALICET C Du tremblement diastolique dans les insuffisances aortiques *J Med Sci* 353 353 July 4 1947
- 69 FRIEDMAN R RICHARD M CALLAGHAN I AND HARTER R Souffles diastoliques anormaux par insuffisance cardiaque fonctionnelle *J Med Sci* 350 353 1949
- 70 FRIEDMAN R TERNAN A CHEN I AND CALLAGHAN I Un nouveau signe de pleurésie cardiaque la vibration proto et métrique *Arch mal coeur* 3 185 1945
- 71 FRIEDMAN I AND STICKERT F C Ueber ein neues Symptom bei Lung und Lungenarterienklappen *Wien klin Wchnchr* 35 200 1902
- 72 FRIED J Occurrence and prognostic significance of gallop rhythm *Acta med Scandinav* 134 153 1949
- 73 FRIED J Phonocardiographic studies on gallop rhythm *Acta med Scandinav* 133 979 1949
- 74 FRIED J AND BAC J Some cases of pre-aortic sound audible at a distance *Acta Med Scandinav* 105 411 1940
- 75 FRIEDMAN R HART C L AND BARTON H C Malignant mediastinal teratoma simulating extracardiac disease *Dis Chest* 27 537 1955

- and fluoroscopic and electrocardiographic studies *J Pediatr* **32** 39 1948
- 432 FRIANGER J A note on the contractility of the musculature of the auriculo ventricular valves *Abstract Am J Physiol* **10** 150 1916
- 433 FRIANGER J Relation of longitudinal tension of an artery to the prearteriotic phenomenon *Am Heart J* **19** 398 1910
- 434 FRIANGER J Studies in blood pressure estimation by indirect methods II Mechanism of compression sounds of Korotkoff *Am J Physiol* **40** 92 1916 III Movement in the artery during pneumatic compression *Am J Physiol* **11** 84 1921
- 435 FUCH I AND HEGGER H Trikuspidalklappenschluss bei a-v Block im Venenpuls *Ztsch Kreislaufforsch* **45** 29 1956
- 436 GATREY H AND DAMPSHIRE W Familial hypoplastic anemia of childhood report of eight cases in two families with beneficial effect of splenectomies in one case *Am J Dis Child* **73** 671 1947
- 437 JTTINGER W Auskultatorische Methode der Blutdruckbestimmung und ihr praktischer Wert *Wien klin Wchschr* **20** 992 1907
- 438 IANNA I AND CURRY R W Dissecting aneurysm of the aorta *South M J* **49** 238 1906
- 439 IANNA W *Cardiology* Butterworth London 1948
- 440 IANNA W Heart murmurs *Brit Heart J* **9** 225 1947
- 441 IANNA W Mitral systolic murmurs *Brit M J* **1** 8 1943
- 442 IANNA W The use of the phonocardiograph in clinical cardiology *Brit Heart J* **10** 92 1948
- 443 IANNA W AND BRAYSON R Max thrombus of the left auricle *Brit Heart J* **10** 39 1948
- 444 IANNA W SHORT D S AND BELFORD D I Solitary pulmonary hypertension *Brit Heart J* **19** 93 1957
- 445 IAWAT W Clinical lecture on heart sounds and on accuracy in cardiac auscultation *Lancet* **1** 1241 1893
- 446 IAWAT W Note on the auscultation of the second sounds of the heart *Lancet* **2** 769 1891
- 447 FAGET J C Notice scientifique sur Roussel de Saint Pons (Hérault) *L'Union Méd* **29** (n.s.) 321-327 337 316 385-395 1866
- 448 FAGET C H On the murmurs attendant upon mitral contraction *Clin Hosp Rep* **16** 217 1871
- 449 FAIR G Acoustics of bronchial breath sounds *Arch Int Med* **39** 286 1927
- 450 FAIRFAX N Anatomical observations on a human body dead of odd diseases as they were communicated by Dr N F Phil Trans Roy Soc Nov 11 1867
- 451 FAIRFAX J Études expérimentales sur les motions organiques du coeur *Gaz méd Paris* **11** 796 1856
- 452 FANCONI
- 453 FARRAR J T AND INGLEFINGER F J Calcium intestinal motility as revealed by study of abdominal sounds *Gastroenterology* **11** 59 1945
- 454 FAUVEL S A biographies of *Lancet* **2** 893 1881 *Car méd Paris* **1** 565-577 1881 *Bull Acad de méd Paris* **8** 160 1884
- 455 FAUVEL S A Mémoire sur les signes stéthoscopiques du rétrécissement de l'orifice auriculo ventriculaire gauche du coeur *Arch gén de méd* **1** 1 1843
- 456 FENSTEIN A R AND DI MASSA R The prognosis of rheumatic endocarditis *American Fed for Clin Research Atlantic City May 4* 1948
- 457 FERENCZ C JOHNSON A I AND WICKESWORTH F W Congenital mitral stenosis *Circulation* **9** 161 1954
- 458 FERRER M I HARVEY H M KUSCHNER M RICHARDS D W JR AND COLMAN A Hemodynamic studies in tricuspid stenosis of rheumatic origin *Circulation Res* **1** 49 1953
- 459 FERTMAN M H AND WOLFE I Calcification of the mitral valve *Am Heart J* **31** 580 1916
- 460 FISCHER J Die auskultatorische Blutdruckmessung im Vergleich mit der oscillatorischen von Heinrich von Recklinghausen und ihr durch die Lungenbestimmung bedingter Ischämischer Wert *Ztschr f physik u diätet Therap* **17** 389 1909
- 461 FISHER J D Observations on cerebral auscultation *Am J Med Sc* **22** 277 1838 (*Arch communtication*) *Med Mag* **2** 144 1834
- 462 FISHER T Diastolic bruit at apex in heart disease of children *Brit M J* **1** 906 1944
- 463 FISCHER B L AND FRIEDLAND C Phonocardiografia en las cardiopatías congénitas *Principia Cardiologica* **4** 285 and 335 1951
- 464 FISHERMAN A P TURNO C M BRANDON BREWER M AND HINSHLWORTH A The pulmonary collateral blood flow in man (Abstract) *J Clin Investigation* **37** 591 1958
- 465 FLAXMAN N Historical aspects of mitral stenosis *Med Life* **45** 3 1978
- 466 FLETCHER H AND MINOW A A Joudes its definition measurement and calculation *J Acou Soc Am* **5** 82 1933
- 467 FINE A On cardiac murmur *Am J Med Sc* **11** 29 1867
- 468 FLINT A On the mitral pre systolic and a mitral diastolic heart murmur *Lancet* **1** 418 1881
- 469 FLINT A Variation in pitch in percussion and respiratory sound and their application to physical diagnosis *Tr A M A* **5** 75 1907
- 470 FOOTE D H The innocent (functional) cardiac murmur in children *Pediatric* **10** 793 1907
- 471 FORBES J Translator's preface to *Lancet's Treatise on the Diseases of the Chest* (first Amer

- 56 GELFAND D The phonocardiogram as a diagnostic aid J Lha's (en Re p 1 45 1930
- 57 GELFAND D and BELLET S The musical murmur of aortic insufficiency clinical muscle examinations based on study of 15 cases Am J M Sc 221 644 1931
- 58 GELFAND R and LEVINE S A The incidence of acute and subacute bacterial endocarditis in congenital heart disease Am J M Sc 204 371 1943
- 59 GELFAND D B and SILBERSTEIN K Perforation of peptic ulcer of the oesophagus into the pericardial cavity Report of a case Brit M J 2 1413 1936
- 60 GENDRY A N Leçons sur les maladies du cœur et des gros vaisseaux p 31 Ballière Paris 194
- 61 GERHARDT C Lehrbuch der Insultation und Perkussion H Lippert Tübingen 1905
- 62 GIBBARD W W On the Diagnosis of Diseases of the Chest based upon the Comparison of Their Physical and General Signs Rev A Little Philadelphia 1936
- 63 GIBSON T B and HARR R I Obstructive brachiocephalic arteritis pulmonic disease of Takayasu Circulation 35 845 1937
- 64 GIBSON G A Diseases of the Heart and Lungs p 61 203 310 317 Young & Kentland Ltd London 1894
- 65 GILLESPIE J A Life of Sir William Tennant Gairdner James Maclehose and Sons Ltd Glasgow 1919
- 66 GILLESPIE J A Lesions of the arterial duct and its clinical significance Edinburgh M J 3 1 1900
- 67 GILLESPIE J A The significance of a histological evidence of valve in the jugular pulse Lancet 2 43 1931
- 68 GISSON R and WILSON J The diagnosis of tricuspid regurgitation Brit Heart J 17 107 1935
- 69 GLENN J L FORTA W J and LANGEBAUM W H Aortic and pulmonary communication due to localized congenital defect of the aortic septum Pediatrics 6 357 1930
- 70 GLENN J M DART T J and HORTON B T Features of coarctation and transverse gradient report of a cardiovascular anomaly with symptoms of tricuspid regurgitation Staff Met Mayo Clin 14 561 1939
- 71 GLOTT G and MULLER C Il valore encefalografico dell'isofono aritografico L 2 a 193
- 72 GLOCKER T R Latent ductus arteriosus and its surgical treatment Brit Heart J 7 1 1935
- 73 GLOCKER T R J and McPHAIL J J Jr Pathologic murmurs in pregnancy a re-emphasis M Ann District of Columbia 24 461 1930
- 74 GLOTT G and BERT J M Recherches cliniques sur l'adynamisme apparent de l'index des sons de la touque au cathétre dans la position d'hyperextension de l'aorte Arch mal coeur 32 51 1939
- 75 GIRARD C CHAPPEL J LATOUR H LECHE I and JEAN R Congenital aortic pulmonary communication Arch mal coeur 48 27 1935
- 76 GITTNER S C Auscultatory blood pressure determinations Arch Int Med 6 196 1910
- 77 GLAZIER S H Concerning the mechanism of production of Korotkoff sounds and their significance in blood pressure determination Bull John Hopkins Hosp 44 174 1909
- 78 GLAZIER A J and THOMSON S The changing heart murmurs of acute rheumatism L Edinburgh M J 11 679 1911
- 79 GLENN J L Fortmeier and telecardiographic Schallphonome Ztschr Kreislaufforsch 22 302 1933
- 80 GLENN J L Zum Problem der Intonation des Herztone Ztschr Kreislaufforsch 11 101 1907
- 81 GLENN J L and GLENN W H Aneurysm of the sinus of Valvula associated with coronary atherosclerosis Radiology 67 416 1937
- 82 GLENN J L and GLENN W H Idiopathic dilatation of the pulmonary artery South African M J 27 320 1933
- 83 GLENN J L GLENN W H and GLENN J L and GLENN J L Simultaneous (combined) ectothermia of the left and right heart Am Heart J 63 3 1931
- 84 GLENN J L GLENN W H and GLENN J L The effect of the Valvula-like mechanism upon the circulation in normal individuals and patients with mitral stenosis Circulation 5 35 1932
- 85 GLENN J L and GLENN T F Tabular data of the velocity and acceleration of high frequency sound in mammalian tissue J Am Phys 23 3 1930
- 86 GLENN J L GLENN J M and GLENN J H Vascular involvement in generalized scleroderma Ills Chet 25 91 1934
- 87 GLENN J L and GLENN J F Functional diastolic murmurs in heart valve enlargement in aortic aneurysm Arch Int Med 60 236 1937
- 88 GLENN J L The heart in (case) diastolic ectothermia 103 37 1930
- 89 GLENN J L GLENN J M and GLENN J H The simultaneous flutter of the diaphragm simulating coronary occlusion J Am M 116 1633 1911
- 90 GLENN J L GLENN J M and GLENN J H Systemic anomalies of the tricuspid valve Am Heart J 45 131 1933
- 91 GLENN J L GLENN J F GLENN J M GLENN J H and GLENN J M Rheumatic tricuspid stenosis Brit M J 2 1351 1937
- 92 GLENN J L Symposium on Cardiovascular Sound Chicago Oct 1934 Circulation in Press
- 93 GLENN J L GLENN J M and GLENN J H Simultaneous pulse pressure in the human left atrium ventricle and aorta Preliminary communication Circulation Re 2 432 1934

- 500 FRY D L MALLOS A J AND CASPER A G T  
A catheter tip method for measurement of the instantaneous aortic blood velocity *Circulation Res* 4 627 1956
- 501 FRY D I NORRIS F W AND MALLOS A J An electric device for instantaneous and continuous computation of aortic blood velocity *Circulation Res* 5 75 1957
- 502 CAD J Klappen-pfeil im Ochsenherzen *Arch f Physiol* p 380 1886
- 503 GADRAT AND MOREAU Thrombose des troncs de la crosse aortique *Arch mal coeur* 45 830 1952
- 504 GAGER L T Dissecting aneurysm of aorta complicating hypertension *Am Heart J* 3 489 1925
- 505 GAIRDNER W T Case of obstruction of the right auriculo ventricular orifice caused by a tumour in the auricle acting as a ball valve etc *Edinburgh Hosp Rep* 1 221 1893
- 506 GAIRDNER W T Short account of cardiac murmurs *Edinburgh M J* 7 438 1861
- 507 GATLIN A L On the interpretation of cardiographic tracing *Clinic Hosp* p Rep 20 280 1875
- 508 GALBRAITH B T AND NORMAN S J Dissecting aneurysm of the aorta *New England J Med* 280 670 1954
- 509 CALEN *Medicorum Graecorum Opera* Latin edition Vol 5 p 444-4 1823
- 510 GALLAVARDIN L À propos de la circulation du rétrécissement mitral L'ultime partie du roulement présystolique ne serait-elle pas protosystolique? *J méd Lyon* 33 991-994 1952
- 511 GALLAVARDIN L Contraction auriculaire perceptible à l'oreille dans le bloc total *Arch mal coeur* 7 171 1914
- 512 GALLAVARDIN L *Lyon méd* 105 401 1905 Cited by Morgagnie and Wolferth (1940)
- 513 GALLAVARDIN L Note sur le rythme de ventilation du souffle anémique de la base et son explication par le reflux protodiastolique du sang de l'artère pulmonaire dans l'infundibulum *Arch mal coeur* 1 421 1908
- 514 GALLAVARDIN L Nouvelle observation avec un topie d'un pseudo-dédoublement du deuxième bruit du coeur simulé par le dédoublement mitral par bruit extracardiaque télé-systolique urajouté *Prat méd franc* 13 19 1932
- 515 GALLAVARDIN L L'endo-dédoublement du deuxième bruit simulé par le rétrécissement mitral par bruit extracardiaque télé-systolique urajouté *Lyon méd* 121 409 1913
- 516 GALLAVARDIN L Rétrécissement aortique avec maximum du souffle dans le cinquième espace intercostal gauche et la région endopexienne *Lyon méd* p 382, March 23 1924
- 517 GALLAVARDIN L AND BERNHEIM M Bruit musical diastolique à apparition brusque et intermittent dans l'insuffisance aortique *Lyon méd* 135 64 1924
- 518 CALLASARDIN I AND BEUTTER C Souffle cardio pulmonaire diastolique de la base du coeur avec autopsie Inspiration à rythme diastolique *Lyon méd* p 1106 June 3 1906
- 519 CALLAVARDIN L AND DELAHAYE J P Ictaphonocardiographie pré et post opératoire dans la sténose mitrale *Cardiologia* 20 90 1957
- 520 CALLAVARDIN L GRASVIER I AND PIERRE R Bruit musical continué chez un cirrhotique *Lyon méd* 142 374 1925
- 521 GALLAVARDIN I AND PALPES RAVAILL Le souffle du rétrécissement aortique peut changer de timbre et devenir musical dans sa propagation apexienne *Lyon méd* p 523 1925
- 522 GALLAVARDIN I AND PALPES RAVAILL Nouveau cas de rétrécissement aortique avec maximum endopexien du souffle *Lyon méd* p 376 Sept 13 1925
- 523 GALLAVARDIN L AND VERT P Nouveau cas de sténotose diastolique par troubles de conduction auriculo ventriculaire *Arch mal coeur* 10 797 1926
- 524 GARRS S The relationship of blood viscosity to the intensity of heart murmurs *Am Heart J* 28 568 1944
- 525 CARDNER F AND ORAM D Intersect left upper ventricle draining pulmonary vein *Brit Heart J* 15 305-316 1953
- 526 GARVIN I F Functional aortic insufficiency *Ann Int Med* 13 1799 1940
- 527 GIBBS B M DILLON R F AND VELA V Further observation of the natural course of ventricular septal defects *New clinical and physiological data Abstract Circulation* 16 885 1957
- 528 GIBBS B M DILLON R F VELA V AND HAIR G Ventricular septal defects Their natural transformation into the a with infundibular stenosis or into the cyanotic or noncyanotic type of tetralogy of Fallot *J A M A* 164 547 1957
- 529 GIBBS B M FELL I H AND CASAS R The diagnosis of aortic septal defect by retrograde aortography *Circulation* 4 251 1951
- 530 GIBBS B M Peroral communication
- 531 GIBBS B M LUKOFF W AND MASON D Teaching auscultation by candle loop type recording *J A M A* 162 1335 1953
- 532 GIBBS B M LUKOFF W MASON D RIEZ R R AND WIRTH C H Cardio-pectrogram *Am Heart J* 48 159 1954
- 533 GIBBS B M MARINO D J AND CREORY J E Early perforation of intraventricular septum after myocardial infarction *J A M A* 148 1413 1952
- 534 CLEI S Auscultation and Percussion Together with the other Methods of Physical Examination of the Chest 1 ed p 143 Smith Elder & Co London 1893
- 535 GEIGER Quoted by Rie (1875)

- 26 GELFAND D. The phonocardiogram as a diagnostic aid. *J Indus Gen Hosp* 1: 43 1950
- 27 GELFAND D and BELLET S. The musical murmur of aortic insufficiency: clinical manifestation. *Heart* 19: 15-24. *Am J M Sc* 221: 644 1951
- 28 GELFAND R and LEVINE S A. The incidence of acute and subacute bacterial endocarditis in congenital heart disease. *Am J M Sc* 204: 31 1953
- 29 GELFAND D D and SILBERTEIN H. Perforation of peptic ulcer of the stomach into the pericardial cavity. Report of a case. *Brit M J* 2: 1113 1956
- 30 GENDRY A N. *Leçons sur les maladies du cœur et de la circulation* p 31. Ballière Jan 1917
- 31 GERHARDT C. *Lehrbuch der Halskrankheiten und Perkussion*. H. Laupp, Tübingen 1916
- 32 GERHARDT W W. On the Diagnosis of Diseases of the Chest. Based upon the Comparison of Their Physical and General Signs. *Key* C. Biddle Philadelphia 1936
- 33 GIBBS T R and KING R I. Multiterative brachiocephalic arteritis: pulmonary disease of Takayasu. *Circulation* 15: 45 1957
- 34 GIBSON C A. Diseases of the Heart and Aorta. pp 61 303 310-317. Young J. Sefton and Edinburgh 1919
- 35 GIBSON C A. *A Life of Sir William Tennant Gifford*. James Maclehose and Son (Ld) Ltd 1912
- 36 GIBSON C A. Lesion of the arterial duct and its diagnosis. *Edinburgh M J* 8: 1 1900
- 37 GIBSON C A. The significance of a hitherto undescribed wave in the jugular pulse. *Lancet* 2: 350 1917
- 38 GIBSON J and WILSON P. The diagnosis of stenopulmonary. *Brit Heart J* 17: 557 1955
- 39 GIBSON S, LOTT W J and LANCHESTER W H. Aortic and pulmonary communication due to localized congenital defect of the aortic septum. *Pediatrics* 6: 35 1950
- 40 GIFFIN H M, DRY T J and HERTZ B T. Reversed correlation and aomot gradient report of a cardiovascular anomaly with symptom of brain tumor. *Proc Staff Meet Mayo Clin* 14: 561 1939
- 41 GIGLI G and MIESAN C. Il valore semeiologico della fonocardiografia. *Is* 1950
- 42 GILCHRIST A R. Patent ductus arteriosus and its surgical treatment. *Brit Heart J* 7: 1 1945
- 43 GILSON R J and McPHERSON J J Jr. Physiological murmurs in pregnancy: a re-emphasis. *M Ann D (Sect of Columbia)* 46: 1 1950
- 44 GIRAUD G and BERT J M. Recherches cliniques sur l'aliment apparent de l'index tonnel. *diastolique par auscultatoire dans la position d'hyperextension de l'avant-bras*. *Arch mal coeur* 32: 57 1939
- 45 GIRAUD C, CHAPTEL J, LATOUR H, LIECHT and JEAN R. Congenital aortic pulmonary communication. *Arch mal coeur* 48: 27 1955
- 46 GITTENES S C. An eulterior blood pressure determination. *Arch Int Med* 6: 196 1910
- 47 GLADSTONE S H. Concerning the mechanism of production of the aortic sound and their significance in blood pressure determination. *Bull John Hopkin Hosp* 44: 127 1929
- 48 GLASSBROOK A J and THOMSON S. The changing heart murmurs of acute rheumatism. *Edinburgh M J* 69: 1911
- 49 GLAUER F. Über die auscultatorische Selbphilphonomen. *Ztschr Herz lausforsch* 42: 123 1933
- 50 GLAUER F. Zum Problem der Intentionale Herztöne. *Ztschr Herz lausforsch* 41: 17 1932
- 51 GLENN A V and CRAMER W H. Aneurysm of the sinus of Valves associated with calcification. *Radiology* 67: 416 1956
- 52 GLENN R H and NELSON M. Idiopathic dilatation of the pulmonary artery. *South African M J* 27: 360 1953
- 53 GLIERBERG H, DICKEY J, RABER C and HARTZ F Jr. Simultaneous (combined) enlargement of the left and right heart. *Am Heart J* 22: 57 1921
- 54 GLIERBERG H, GLIERBERG I I and HARTZ F N. The effects of the Valves like maneuver upon the circulation in normal in lard and in relation with mitral stenosis. *Circulation* 5: 38 1951
- 55 GLIMAN D I and HARTZ T F. Talular late of the velocity and relation of high frequency sound in mammalian tissue. *J Acoust Soc Am* 28: 35 1956
- 56 GLIMAN R, YOUNG J M and KNOX F H. Myocardial involvement in generalized scleroderma. *Dis Chest* 22: 91 1954
- 57 GLIMAN H and BIRN I I. Functional diastolic murmurs and left heart enlargement in severe anemia. *Arch Int Med* 89: 296 1959
- 58 GLIMAN J S. The heart in Graves disease. *Practitioner* 105: 37 1950
- 59 GLIMAN J M. Paroxysmal flutter of the diaphragm stimulating coronary occlusion. *J A M A* 116: 1633 1911
- 60 GLIMAN J F, WYNN A and STEINER R I. Isthmian anomaly of the tricuspid valve. *Am Heart J* 45: 141 1953
- 61 GLIMAN J F, RICE S M, SIMON A H and FLOW M. Rheumatic tricuspid stenosis. *Brit M J* 2: 1353 1957
- 62 GLIMAN J. Symposium on Cardiovascular. *Chicago Oct 2, 1955*. *Circulation* 11: 1955
- 63 GLIMAN A J, BRADSHAW F and RAVITCH M M. Simultaneous pre- and post- in the human left atrium, ventricle and aorta. *Arterial communication*. *Circulation* 12: 475 1955

- 572 GORDON A J, GEMMINS C, FRISHMAN A AND NABATOFF R A. Tricuspid stenosis. Report of a case with hemodynamic studies at tricuspid commissurotomy. *Am J Med* 22 306 1957
- 573 GORDON W. Posture and heart murmurs. *Brit M J* 1 636 1902
- 574 GORLIN R. In Second Symposium on Cardiovascular Sound Circulation in press
- 575 GORLIN R AND GORLIN S C. Hydraulic formula for calculation of area of stenotic mitral valve, other cardiac valve, and central circulatory shunts. *Am Heart J* 41 1 1951
- 576 GORLIN R, HAYNES I W, GOODALE W T, SAWYER C C, DOW J W AND DEXTER I. Studies on the circulatory dynamic in mitral stenosis. II. Altered dynamics at rest. *Am Heart J* 41 30 1951
- 577 GORLIN R, KNOWLES J H AND STOREY C I. The Valvular maneuver as a test of cardiac function. I. Pathologic physiology and clinical significance. *Am J Med* 22 107 1957
- 578 GORLIN R, McVILLAN J K, MLEDD W E, MATTHEWS M B AND DALEY R. Dynamics of the circulation in aortic valvular disease. *Am J Med* 18 855 1955
- 579 GORLIN R, SAWYER C C, HAYNES I W, GOODALE W T AND DEXTER I. Effects of exercise on circulatory dynamics in mitral stenosis. III. *Am Heart J* 41 192 1951
- 580 GOLD S L. ed. *Pathology of the Heart* C C Thomas Springfield 1953
- 581 GOSLEY B A. The aortic valvular lesion associated with Austin Flint murmur. *Am Heart J* 22 208 1941
- 582 GRAY W, MÖLLER F AND MANNHEIMER I. The continuous murmur. Incidence and characteristics in different parts of the human body. *Acta med Scandinavica Suppl* 196 167 1947
- 583 GRANT R P. Architectonics of the heart. *Am Heart J* 45 40 1953
- 584 GRANT R P. A precordial systolic murmur of extracardiac origin during pregnancy. *Am Heart J* 52 944 1956
- 585 GRATER H AND HEGGER R. Experimentelle Untersuchungen über die energetischen dynamische Herzinsuffizienz. *Cardiologia* 11 1 1948
- 586 GRAVES R J. Clinical lectures. London Med & Surg J 7 516 1835
- 587 GRAY I R. Paradoxical splitting of the second heart sound. *Brit Heart J* 18 21 1956
- 588 GRAY J. *History of the Royal Medical Society* 1737 1937 p 91 University Press Edinburgh 1952
- 589 GRAYBRIEL A AND GLENDY R F. Circulatory effects following the intravenous administration of pitressin in normal persons and in patients with hypertension and angina pectoris. *Am Heart J* 21 481 1941
- 590 GREENE D G, BALDWIN E DE F, BALDWIN J S, HIMMELSTEIN A, ROH C L AND CHURCHAND A. Pure congenital pulmonary stenosis and idiopathic congenital dilatation of pulmonary artery. *Am J Med* 6 24 1919
- 591 GREENE D G AND BUNNELI I J. The circulatory response to the Valvular maneuver of patients with mitral stenosis with and without autonomic blockade. *Circulation* 8 264 1953
- 592 GREENE J A. Unusual sounds emanating from the chest. *Arch Int Med* 71 410 1913
- 593 GRIFFITH I J AND PROCTOR V S F. Detecting aneurysm of the aorta with repeated intrapericardial hemorrhages. *Clin Proc* 7 21 1918
- 594 GRELAND R. Perforation of the infarcted interventricular septum diagnosed ante mortem. *Acta med Scandinavica* 133 1 1949
- 595 GRESHAM C A. Networks on the right side of the heart. *Brit Heart J* 18 391 1957
- 596 GRIFFITH C C AND VITALE I L. Acute and subacute disseminated lupus erythematosus. A correlation of clinical and postmortem findings in 15 cases. *Circulation* 3 492 1951
- 597 GRIFFITH I P. Mid-systolic and late systolic mitral murmurs. *Am J Med Sci* 104 785 1902
- 598 GRIFFITH T W. Remarks on two cases of heart block. *Heart* 3 143 1912
- 599 GRISHMAN A, KROOP I C, STEINBERG M F AND DACK S. Pre-systolic pulsations of liver in absence of tricuspid disease. *Am Heart J* 40 731 1950
- 600 GROFDEI I M. *The Venous Pulse and its Graphic Recording*. Brooklyn Med Press New York 1946
- 601 GROFDEI F M AND BORCHARDT I R. Heart murmurs recorded intrathoracically. *Exper Med & Surg* 9 144 1951
- 602 GROFDEI I M AND MILLER M. Graphic study of aortic blood pressure measurement. *Exper Med & Surg* 1 148 1913
- 603 GROFDEI F M AND MILLER M. The influence of the chest wall on the heart sounds. *Exper Med & Surg* 2 325 1914
- 604 GROFDEI F M AND MILLER M. Intratracheal intubation. *Exper Med & Surg* 6 49 1950
- 605 GROFDEI F M AND MILLER M. Studies on the acoustic phenomena over the vessels of the neck in the healthy and the diseased heart. *Exper Med & Surg* 11 193 1944
- 606 GROFDEI F M AND MILLER M. The nature and origin of the so-called systolic gallop rhythm. *Exper Med & Surg* 3 107 1945
- 607 GROOM D. Second symposium on cardiovascular sound. *Circulation in press*
- 608 GROOM D AND BOONE J A. The dove coo murmur and murmurs heard at a distance from the chest wall. *Ann Int Med* 42 1214 1953
- 609 GROOM D AND BOONE J A. The recording of heart sounds and vibrations. II. The applica-

- tion of an electronic pickup in the graphic recording of ultrahigh and audible frequencies *Exper Med & Surg* 14: 233 1946
- 68 GRAY D BUCK J A AND JENKINS M A A human cardiac auscultation J A M A 199: 649 1950
- 69 CROOK D HERRING O FRANK W AND SHEELY G The effect of background noise on cardiac auscultation *Am Heart J* 52: 741 1956
- 70 CROOK D AND SIMONSON J T A high sensitivity pickup for echocardiography *Am Heart J* 51: 507 1956
- 71 GRAY D LADENBERG A F BIDWELL J B AND LADENBERG I The recording of heart sound and vibration I Theoretical review and description of a new electronic direct contact vibration pickup *Exper Med & Surg* 14: 229 1946
- 72 GRAY I Scalloped congenital bicuspid aortic valve *Arch Path* 23: 340 1953
- 73 GRAY I E Surgical closure of an aortic septal defect *Circulation* 5: 508 1952
- 74 GREENBERG H O The cardiac complication of ankylostoma infection with special reference to a presystolic murmur occurring in the chest *J Trop Med* 36: 39 1953
- 75 GUN A L AND WILSON M C The amplification and recording of fetal heart sound *Proc Roy Soc Med* 46: 9 1953
- 76 GRAY J A An unusual case of a continuous murmur imitating patent ductus arteriosus and associated with other congenital cardiac defects *Thorax* 12: 31 1956
- 77 GUYTON T C AND WICKER C J Baroreceptor name change produced by aortic resection of different degree *Circulation* 5: 133 1952
- 78 LITTMAN J *Handbook of Physical Diagnosis* Tenth Edition for the New Sydenham Society 1959
- 79 LITTMAN J Pneumopericardium entsteht durch Perforation eines runden Magen Lochs in den Herzbeutel *Berl klin Wochenschr* 1950
- 80 LITTMAN J Über den geräuschlosen stillen Herzton bei der Stenose der Aortenklappen *Arch f path Anat* 46: 105 1956
- 81 GUN N B Congenitally murmurs found in hepatic cirrhosis and their confusion with murmurs of congenital heart disease *Am J M S* 180: 575 1950
- 82 LILLENWALD C LUDWIG W F ANDERSON A AND MOFFET T Congenital multiple peripheral stenoses of the pulmonary artery branches *Circulation* 19: 399 1959
- 83 HAYES BRUCE R AND ADAMS R The duration of ventricular response mechanically and electrically recorded as influenced by rate, tension and fatigue *Am J Physiol* 87: 76 1928
- 84 HALL J J McDONALD B A AND WOODLEY J R Velocity profile of oscillating arterial flow with some calculation of viscous drag and the Reynold's number *J Physiol* 125: 11 1955
- 85 HAYS M R AND LIEBOW A A Cysticercal calcification to the lung in congenital pulmonary stenosis *Bull Internat A M M* 25: 1 1948
- 86 HALL J N Late systolic mitral murmur *Am J M S* 125: 673 1953
- 87 HANBY W B AND TRACY R N Air embolism in operations done in sitting position *Surg Gynecol* 31: 26 1922
- 88 HANSEN K I Heart murmur *Am J M S* 131: 16 1951
- 89 HAMILTON C R COTTE R J AND JENKINS A Arteriovenous fistula of renal vessel Case report and review of literature *J Urol* 62: 203 1957
- 90 HANMAN I Clinical pathological conference *Internat Clinic (Hrsgen)* 2: 1 1953
- 91 HANMAN I Diagnosis of the causes of heart failure *New England J Med* 219: 28 1958
- 92 HANMAN I Mediastinal emphysema *J A M A* 126: 131 1951
- 93 HANMAN I Spontaneous interstitial emphysema of the lung *Tr A Am Physician* 17: 311 1952
- 94 HANMAN I Spontaneous mediastinal emphysema *Bull J Clin Hosp* 64: 1 1957
- 95 HANMAN I AND HANSHAW W F Juvenile hypertrophic cardiomyopathy *Bull J Clin Hosp* 67: 71 1953
- 96 HANMAN I AND RICH A R Clinical pathologic conference—tw cases of subacute bacterial endocarditis *Internat Clin* 2: 201 1953
- 97 HANSEN C W MCDONALD B M JR AND STAMM M H ANDERSON W H AND STAMM C W B Aortic stenosis of no physiologic significance *New England J Med* 258: 305 1958
- 98 HANSEN C W ANDERSON W H AND STAMM C W B Aortic stenosis of the aortic valve *Artif Circulation* 18: 101 1953
- 99 HANSEN A T Free wire measurement in the human organ *Acta Physiol Scandinavica (Suppl)* 19: 1 30 1953
- 100 HANSEN A T AND WICKER C I A new technique of differential free wire measurements using condenser microphones *Acta Physiol Scandinavica* 22: 211 1950
- 101 HANSEN C M MCDONALD B M AND COTTE R J A Cinecardiography in patent ductus arteriosus *Circulation* 10: 301 1954
- 102 HARRIS J D SM Relations between vision and audition *Charles C Thomas Springfield Ill* 1950
- 103 HARRIS T N Phonocardiographic study of pulmonary stenosis murmurs in children *Am Heart J* 60: 805 1955



- 572 CORDON A J GENAINS G FRISHMAN A AND VABATOFF R A Tricuspid stenosis Report of a case with hemodynamic studies at tricuspid commissurotomy Am J Med 22 306 1957
- 573 GORDON W Posture and heart murmurs Brit M J 1 636 1902
- 574 GORLIN R In Second Symposium on Cardiovascular Sound Circulation in press
- 575 GORLIN R AND GORLIN S G Hydraulic formula for calculation of area of stenotic mitral valve other cardiac valves and central circulatory shunts Am Heart J 41 1 1951
- 576 GORLIN R HAYNES F W GOODALE W T SAWYER C G DOW J W AND DEXTER I Studies on the circulatory dynamic in mitral stenosis II Altered dynamics at rest Am Heart J 41 30 1951
- 577 GORLIN R KNOWLES J H AND STOREY C F The Valsalva maneuver as a test of cardiac function Pathologic physiology and clinical significance Am J Med 22 197 1957
- 578 GORLIN R McMILLAN I K MIDD W E MATTHEWS M B AND DALEY R Dynamics of the circulation in aortic valvular disease Am J Med 18 855 1955
- 579 GORLIN R SAWYER C G HAYNES F W GOODALE W T AND DEXTER I Effects of exercise on circulatory dynamic in mitral stenosis III Am Heart J 41 192 1951
- 580 COLD S I ed Pathology of the Heart C C Thomas Springfield 1953
- 581 COLEY B A The aortic valvular lesion associated with Austin Flint murmur Am Heart J 22 208 1941
- 582 CRAWFORD MOLLER F AND MANNHIMER F The continuous murmur Incidence and characteristic in different parts of the human body Acta med Scandinav Suppl 196 167 1947
- 583 CRANT R P Architectonics of the heart Am Heart J 46 40a 1953
- 584 CRANT R P A precordial systolic murmur of extra-cardiac origin during pregnancy Am Heart J 62 944 1956
- 585 CRAIGER H AND HEGGLIN R Experimentelle Untersuchungen über die sog energetisch dynamische Herzinsuffizienz Cardiologia 11 1 1948
- 586 GRAVES R J Clinical lectures London Med & Surg J 7 516 1835
- 587 GRAY I R Paradoxical splitting of the second heart sound Brit Heart J 18 21 1956
- 588 GRAY J History of the Royal Medical Society 1737-1937 p 91 University Press Edinburgh 1952
- 588A GRAYBIE A AND GLENDY R I Circulatory effects following the intravenous administration of pitressin in normal persons and in patients with hypertension and angina pectoris Am Heart J 21 481 1941
- 589 GREENE D G BALDWIN E DE F BALDWIN J S HILFSTEIN A ROH C L AND COLEMAN A Pure congenital pulmonary stenosis and idiopathic congenital dilatation of pulmonary artery Am J Med 5 24 1949
- 590 GREENE D C AND BUNNEY I I The circulatory response to the Valsalva maneuver of patients with mitral stenosis with and without autonomic blockade Circulation 8 264 1953
- 591 GREENE J A Unusual sounds emanating from the chest Arch Int Med 71 410 1943
- 592 GREK I J AND PROCTOR N S F Dissecting aneurysm of the aorta with repeated intrapericardial hemorrhages Clin Proc 7 241 1945
- 593 GRIELAND R Perforation of the infarcted interventricular septum diagnosed ante mortem Acta med Scandinav 133 1 1949
- 594 GRESHAM G A Network on the right side of the heart Brit Heart J 19 381 1957
- 595 GRIFFITH G C AND VURAL I L Acute and subacute disseminated lupus erythematosus A correlation of clinical and postmortem findings in 19 cases Circulation 8 402 1951
- 596 GRIFFITH J P Mid-systolic and late systolic mitral murmurs Am J M Sc 104 285 1952
- 597 GRIFFITH T W Remarks on two cases of heart block Heart 3 143 1912
- 598 GRISHMAN A KROOP I G STEINBERG M F AND DACK S Pre-systolic pulsations of liver in absence of tricuspid disease Am Heart J 40 731 1950
- 599 GROEDFEL F M The Venous Pulse and its Graphic Recording Brooklyn Med Press New York 1946
- 600 GROEDFEL F M AND BORCHARDT P R Heart murmurs recorded intrathoracically Laper Med & Surg 9 144 1951
- 601 GROEDFEL F M AND MILLER M Graphic study of aortic blood pressure measurements Exper Med & Surg 1 145 1943
- 602 GROEDFEL F M AND MILLER M The influence of the chest wall on the heart sounds Exper Med & Surg 2 378 1944
- 603 GROEDFEL F M AND MILLER M Intratracheal aortic cathulation Exper Med & Surg 8 42 1950
- 604 GROEDFEL F M AND MILLER M Studies on the acoustic phenomena over the vessels of the neck in the healthy and the diseased heart Exper Med & Surg 2 193 1944
- 604A GROEDFEL F M AND MILLER M The nature and origin of the so-called systolic gallop rhythm Exper Med & Surg 3 107 1945
- 605 GROOM D Second symposium on cardiovascular Circulation in press
- 606 GROOM D AND BOONF J A The dove coo murmur and murmur heard at a distance from the chest wall Ann Int Med 42 1214 1955
- 607 GROOM D AND BOONF J A The recording of heart sounds and vibrations II The applica

- tion of an electronic pickup in the graphic recording of abdominal and audible frequency  
*Exper Med & Surg* 11: 200 1956
- 58 CROOK D, BRYAN J A AND JENNINS M. Venous hum in cardiac auscultation. *J A M A* 159: 631 1955
- 59 CROOK D, HERRING O, FRANCIS W AND SEALEY C. The effect of background noise on cardiac auscultation. *Am Heart J* 51: 195 1956
- 60 CROOK D AND SIMONSEN V T. A high sensitivity pickup for cardiovascular sounds. *Am Heart J* 51: 297 1956
- 61 CROOK D, LINDERBERG A F, BIRWELL J B AND LINDERBERG F. The recording of heart sound and vibration. I. Historical review and description of a new electronic direct contact vibration pickup. *Exper Med & Surg* 14: 279 1956
- 62 CROOK D. So-called congenital bicuspid aortic valve. *Arch Path* 51: 300 1957
- 63 CROOK D. Surgical closure of an aortic aortic defect. *Circulation* 15: 100 1957
- 64 CROOK D AND HERRING O. The earliest complication of ankylosis infection with special reference to aortic valve murmur occurring in the early years. *J Trop Med* 38: 45 1953
- 65 CRYAN A J AND WOOD M C. The significance and recording of fetal heart sound. *Ann Roy Soc Med* 48: 50 1955
- 66 CRYAN A J. Anomalous course of aortic valve murmur imitating patent ductus arteriosus and a coarctation with other congenital cardiovascular defects. *Thorax* 12: 31 1956
- 67 CRYAN A J AND WOOD M C. Bicuspid aortic valve change produced by aortic stenosis at different degrees. *Circulation* 13: 195 1956
- 68 CRYAN A J. *Handbook of Physical Diagnosis*. Translated for the New England Society, 1959
- 69 CRYAN A J. Pneumopericardium. *Internat Arch Exploration eine neuen Vagang chismus in den Herzbeutel*. Berl klin Wochenschr 91: 1950
- 70 CRYAN A J. Über den pathologischen Zustand des Herzens bei der Stenose der Aorta. *Internat Arch Exploration eine neuen Vagang chismus in den Herzbeutel*. Berl klin Wochenschr 91: 1950
- 71 CRYAN A J. On the venous murmur found in hepatic cirrhosis and its relationship with murmur of congenital heart disease. *Am J M* 180: 575 1955
- 72 CRYAN A J, LINDERBERG A F AND LINDERBERG A. Congenital multiple peripheral stenoses of the pulmonary arteries. *Leibniz* 19: 259 1955
- 73 CRYAN A J AND LINDERBERG A. The duration of ventricular response mechanically and electrically recorded as influenced by rate initial tension and fatigue. *Am J Physiol* 87: 70 1955
- 74 HALL J F, McDONALD D A AND WILKINSON J R. Velocity profile of oscillating arterial flow with some calculation of viscous drag and the Reynold number. *J Physiol* 125: 679 1955
- 75 HALL J F AND LINDERBERG A. Collateral circulation to the lung in congenital pulmonary stenosis. *Bull Internat A M M* 1: 1955
- 76 HALL J F. Late aortic mitral murmurs. *Am J M* 180: 125 1955
- 77 HANSEN W B AND TERRY R N. Air embolism in operations and in sitting position. *Surgery* 51: 71 1955
- 78 HANSEN W B. Heart murmur. *Am J M* 180: 181 1955
- 79 HAMILTON C R, CRYAN A J AND JENNINS M. Arteriovenous fistula of renal vessels. Case report and review of literature. *J Urol* 69: 283 1953
- 80 HANSEN W B. Clinical pathologic conference. *Internat Clin* 4 (1st series): 251 1953
- 81 HANSEN W B. Diagnosis of the early stage of heart failure. *New England J Med* 215: 285 1955
- 82 HANSEN W B. Media tinal embolism. *J A M A* 155: 125 1955
- 83 HANSEN W B. Spontaneous inter tinal embolism of the lung. *Tr A Am Phys Soc* 82: 211 1955
- 84 HANSEN W B. Spontaneous media tinal embolism. *Bull John H Hopk Hosp* 1: 1955
- 85 HANSEN W B AND LINDERBERG A. In subacute infectious viral septemia. *Bull John H Hopk Hosp* 51: 113 1955
- 86 HANSEN W B AND LINDERBERG A. Clinical pathologic conference. Case of subacute bacterial endocarditis. *Internat Clin* 2: 201 1953
- 87 HANSEN W B, LINDERBERG A AND LINDERBERG A. Aortic stenosis of posthypertensive significance. *New England J Med* 258: 700 1958
- 88 HANSEN W B. The aortic valve at tract. *Circulation* 11: 591 1957
- 89 HANSEN W B. The use of measurement in the human organism. *Acta Physiol Scandinav* (supp 68): 19 1951
- 90 HANSEN W B AND LINDERBERG A. A new technique of differential pressure measurement using condenser manometers. *Acta Physiol Scandinav* 22: 211 1951
- 91 HANSEN W B, LINDERBERG A AND LINDERBERG A. Thrombography in patent ductus arteriosus. *Circulation* 10: 501 1954
- 92 HANSEN W B. Some of the relations between vision and color. *Charles C Thomas* (Springfield) Ill 1950
- 93 HANSEN W B. Phonocardiographic study of pulmonary aortic murmurs in children. *Am Heart J* 50: 605 1955

- 572 GORDON A J GENAIN, C FRISHMAN A AND VARATOFF R A Tricuspid stenosis: Report of a case with hemodynamic studies at tricuspid commissurotomy. *Am J Med* 22 306 1957
- 573 GORDON W Posture and heart murmurs. *Brit M J* 1 636 1952
- 574 GORLIN R In Second Symposium on Cardiovascular Sound Circulation in press
- 575 GORLIN R AND GORLIN S C Hydraulic formula for calculation of area of stenotic mitral valve: other cardiac valves and central circulatory shunt. *Am Heart J* 41 1 1951
- 576 GORLIN R HAYNES F W GODDART W T SAWYER C C DOW J W AND DEXTER I Studies on the circulatory dynamic in mitral stenosis II Altered dynamics at rest. *Am Heart J* 41 30 1951
- 577 GORLIN R KNOWLES J H AND STOREY C F The Valvular maneuver as a test of cardiac function: I Pathologic physiology and clinical significance. *Am J Med* 23 197 1957
- 578 GORLIN R McVILLIAN I K MIDG W I MATTHEWS M B AND DALEY R Dynamics of the circulation in aortic valvular disease. *Am J Med* 18 855 1955
- 579 GORLIN R SAWYER C C HAYNES F W GODDART W T AND DEXTER I Effects of exercise on circulatory dynamics in mitral stenosis III. *Am Heart J* 41 192 1951
- 580 COLD E I ed. *Pathology of the Heart* C C Thomas Springfield 1953
- 581 COLLEY B A The aortic valvular lesion associated with Austin Flint murmur. *Am Heart J* 22 208 1941
- 582 RAF W MÖLLER F AND MANNHEIMER I The continuous murmur: Incidence and characteristics in different parts of the human body. *Acta med Scandinav Suppl* 196 167 1947
- 583 CRANT R P Architectonics of the heart. *Am Heart J* 46 405 1953
- 584 CRANT R P A precordial systolic murmur of extracardiac origin during pregnancy. *Am Heart J* 22 944 1956
- 585 CRALER H AND HECCLIN R Experimentelle Untersuchungen über die sog energetisch dynamische Herzinsuffizienz. *Cardiologia* 22 1 1948
- 586 GRAVES R J Clinical lecture. London Med & Surg J 7 516 1835
- 587 GRAY I R Paradoxical splitting of the second heart sound. *Brit Heart J* 13 21 1956
- 588 GRAY J *History of the Royal Medical Society 1737-1937* p 91 University Press Edinburgh 1952
- 589 GRAYBIEL A AND GLENDY R E Circulatory effects following the intravenous administration of pitressin in normal persons and in patients with hypertension and angina pectoris. *Am Heart J* 21 481 1941
- 590 CREENE D C BALDWIN L DE F BALDWIN J S HIMMELSTEIN A ROH C L AND COLEMAN A Pure congenital pulmonary stenosis and idiopathic congenital dilatation of pulmonary artery. *Am J Med* 6 24 1949
- 591 CREENE D C AND BLUNELL I J The circulatory response to the Valsalva maneuver of patients with mitral stenosis with and without autonomic blockade. *Circulation* 8 264 1953
- 592 CREENE J A Unusual sounds emanating from the chest. *Arch Int Med* 71 410 1943
- 593 CREKE I J AND PROCTOR N S F Dissecting aneurysm of the aorta with repeated intracerebral hemorrhages. *Clin Proc* 7 241 1948
- 594 GRIFFLAND R Perforation of the infarcted intraventricular septum diagnosed ante mortem. *Acta med Scandinav* 133 1 1949
- 595 CRISHAM C A Networks on the right side of the heart. *Brit Heart J* 19 381 1957
- 596 CRIFTHITH C C AND VURAL I J Acute and subacute dissecting aortic aneurysm: A correlation of clinical and postmortem findings in 18 cases. *Circulation* 3 492 1951
- 597 CRIFTHITH J P Mid-systolic and late systolic mitral murmurs. *Am J Med Sci* 104 285 1952
- 598 CRIFTHITH T W Remarks on two cases of heart block. *Heart* 3 143 1912
- 599 CRISHMAN A AND ROOF I C STEINBERG M F AND DACK S Free systolic pulsation of liver in absence of tricuspid disease. *Am Heart J* 40 731 1950
- 600 CRODFEL F M *The Venous Pulse and its Graphic Recording*. Brooklyn Med Press New York 1946
- 601 CRODFEL F M AND BORCHARDT P R Heart murmur recorded intrathoracically. *Exper Med & Surg* 9 144 1951
- 602 CRODFEL F M AND MILLER M Graphic study of aortic blood pressure measurement. *Exper Med & Surg* 1 148 1949
- 603 CRODFEL F M AND MILLER M The influence of the chest wall on the heart sound. *Exper Med & Surg* 2 328 1944
- 604 CRODFEL F M AND MILLER M Intrathoracic cultivation. *Exper Med & Surg* 8 47 1950
- 605 CRODFEL F M AND MILLER M Studies on the venous phenomenon over the vessels of the neck in the healthy and the diseased heart. *Exper Med & Surg* 2 193 1944
- 606 CRODFEL F M AND MILLER M The nature and origin of the so-called systolic gallop rhythm. *Exper Med & Surg* 3 107 1945
- 607 CROOM D Second symposium on cardiovascular sound. *Circulation* in press
- 608 CROOM D AND BOONE J A The dove coo murmur and murmurs heard at a distance from the chest wall. *Ann Int Med* 42 1214 1955
- 609 CROOM D AND BOONE J A The recording of heart sounds and vibration II The applica-

- tion of an electronic pickup in the graphic recording of inaudible and audible frequency  
*Exper Med & Surg* 14 350 1956
- 60 GROOM D, BORSE J A and JENNINS M. Venous hum in carotid occlusion. *J A M A* 159 639 1955
- 61 GROOM D, HERRICK O, FRANK W and SHELLEY C. The effect of background noise on carotid occlusion. *Am Heart J* 51 136 1956
- 610 GROOM D and SIMMONS T. A high efficiency pickup for cardiovascular sound. *Am Heart J* 54 579 1957
- 611 GROOM D, LINDERBERG A F, BIDWELL J B and LINDERBERG L. The recording of heart sounds and vibration. I. Historical review and description of a new electronic direct contact vibration pickup. *Exper Med & Surg* 14 237 1956
- 612 GRAY L. So called congenital bicuspid aortic valve. *Arch Path* 23 350 1953
- 613 GRAY R F. Surgical closure of an aortic septal defect. *Circulation* 5 509 1952
- 614 GREENBERG H O. The cardiac complication of ankylostoma infection with special reference to a presystolic murmur occurring in these cases. *J Trop Med* 38 40 1953
- 615 GUN A L and WOOD M C. The amplification and recording of fetal heart sound. *Proc Roy Soc Med* 48 85 1955
- 616 GUNNING J A. An unusual case of a continuous murmur imitating patent ductus arteriosus and associated with other congenital cardiac defects. *Thorax* 12 34 1956
- 617 GUTHRIE T C and WILSON C J. Baric hemodynamic change produced by aortic constriction of different degrees. *Circulation* 3 17 1951
- 618 GUTMAN I. *Handbook of Physical Diagnosis* (Translated) for the New Edinburgh Society 1959
- 619 GUTMAN P. Pneumopericardium entstanen durch Perforation eines runden Magengeschwürs in den Herzbeutel. *Berl Klin Wochenschr* 21 1950
- 620 GUTMAN I. Über dengepulstencharakteristischen Herzton bei der Stenose des thorax aortens. *Arch Path Anat* 48 100 1959
- 621 GUNZ N B. On some venous murmurs found in hepatic cirrhosis and their confusion with murmurs of congenital heart disease. *Am J Med Sc* 180 22 1930
- 622 GYLENHART C, LÖNN H, LINDERBERG A and MÖLLER T. Congenital multiple peripheral stenoses of the pulmonary arteries. *Lebhatte* 19 379 1957
- 623 HAFKE BRING R and AUBAN H. The duration of ventricular repolarization mechanically and electrically recorded as influenced by rate, initial tension and fatigue. *Am J Physiol* 87 300 1955
- 624 HALL J F, McDONALD D A and WOODRUFF J R. Velocity profiles of oscillating arterial flow with some calculation of viscous drag and the Reynold's number. *J Physiol* 128 6 1 1955
- 625 HALL M R and FLEMING A C. Bicuspid aortic valve to the lung in congenital pulmonary stenosis. *Bull Internat A M Mu* 28 1 1954
- 626 HALL J N. Late systolic mitral murmur. *Am J Med Sc* 125 673 1953
- 627 HANBY W B and TERRY R N. Air embolism in operations done in sitting position. *Surgey* 31 21 1952
- 628 HANSEN I. Heart murmurs. *Am J Med Sc* 181 75 1931
- 629 HAMILTON C R, COTTE R J and JENNINS S. Arteriovenous fistula of renal vessels. Case report and review of literature. *J Urol* 69 300 1953
- 630 HANNA L. Clinical pathological conference. Internat Clinic 4 (47th series) 201 1953
- 631 HANNA L. Dissecting aortic aneurysm of heart failure. *New England J Med* 219 250 1958
- 632 HANNA L. Mediastinal emphysema. *Am J Med Sc* 125 1 1955
- 633 HANNA L. Spontaneous interstitial emphysema of the lung. *Tr A Am Physician* 62 311 1957
- 634 HANNA L. Spontaneous mediastinal emphysema. *Bull John Hopkins Hosp* 65 1 1959
- 635 HANNA L and RICHARD W F JR. Subacute streptococcal valvular endocarditis. *Bull John Hopkins Hosp* 67 49 1955
- 636 HANNA L and RICHARD W F JR. Clinical pathological conference—two cases of subacute bacterial endocarditis. *Internat Clin* 2 301 1953
- 637 HANNA L, WILSON W M JR and HANNA L. Aortic stenosis of no pathologic significance. *New England J Med* 258 300 1958
- 638 HANNA L. Endocarditis of the aortic valve. *Internat Circulation* 10 591 1957
- 639 HANNA L. Tissue measurement in the human organism. *Acta physiol Scand* 11 (supp 64) 19 1 230 1959
- 640 HANNA L and WARRICK F. A new technique of differential pressure measurements using condenser manometers. *Acta physiol Scand* 22 211 1960
- 641 HANNA L, HANNA L A and CULBERT M. Phonocardiography in patent ductus arteriosus. *Circulation* 10 501 1954
- 642 HANNA J D. *Some relations between vision and audition*. Charles C Thomas, Springfield, Ill 1950
- 643 HANNA T N. Phonocardiographic study of pulmonary valve murmurs in children. *Am Heart J* 50 800 1955

- 611 HARRIS T N AND IRIFDMAN S Phonocardiographic differentiation of the murmur of mitral insufficiency from some commonly heard adventitious sound in childhood *Pediatrics* 3 845 1949
- 612 HARRIS T N AND IRIFDMAN S Phonocardiographic differentiation of vibratory (functional) murmurs from those of valvular insufficiency further observations and application to the diagnosis of rheumatic heart disease *Am Heart J* 43 707 1952
- 613 HARRIS T N SAITZMAN H A NEEDLEMAN H I AND LISKER I Spectrographic comparison of ranges of vibration frequency among some innocent cardiac murmurs in childhood and some murmurs of valvular insufficiency *Pediatrics* 20 57 1957
- 614 HARRISON I I Correlation of the aorta of the adult type associated with cystic degeneration of the media in the first portion of the arch *Arch Path* 57 742 1939
- 615 HARTMAN F W AND BEHRMANN G Simplified automatic blood pressure recorder Henry Ford Hosp M Bull 1 2 1953
- 616 HARVEY A M AND BORDLEY I III *Differential Diagnosis The Interpretation of Clinical Evidence* W B Saunders Co Philadelphia 1955
- 617 HARVEY A M SHULMAN I L TUMITY I A CONIFF C I AND SCHOFERICH I H Systemic lupus erythematosus review of the literature and clinical analysis of 138 cases *Medicine* 33 291 1954
- 618 HARVEY I N McILROY W D AND WHITNEY A H On cavity formation in water *J Appl Physics* 15 162 1947
- 619 HARVEY I N WHITNEY A H COOPER K W PRAND D C AND McILROY W D The effect of mechanical disturbance on bubble formation in single cells and tissues after saturation with extra high gas pressures *J Cell & Comp Physiol* 23 325 1946
- 620 HARVEY W *Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus* p 49 Translated and annotated by Chuaneet D Leike Chirle C Thomas Springfield 1925
- 621 HARVEY W *Visceral Lectures* 1616 (see front piece of ref 650)
- 622 HARVEY W I In Symposium on Cardiovascular Sound *Circulation* 16 270-290 414-436 1957
- 623 HARVEY W I AND CORRADO M A Multiple sounds in paroxysmal ventricular tachycardia: An aid in diagnosis by auscultation *New England J Med* 257 376 1957
- 624 HARVEY W P AND CORRADO M A Prognosis and classification of gallop rhythm *Abstract Circulation* 14 950 1956
- 625 HARVEY W P AND ELIYEV S A The changing intensity of the first sound in auricular flutter in aid to the diagnosis by auscultation *Am Heart J* 35 924 1918
- 626 HARVEY W P AND SEGAL I P Don't forget your stethoscope *Gen Practitioner* 12 90 1950
- 627 HAYES F Z JR AND GAMBILL I I Cruveilhier Baumgarten syndrome report of a case *Gastroenterology* 21 160 1952
- 628 HAYS I B AND HOGAN W H Rupture of aortic valve cusp attachment due to cystic medial necrosis of the aorta: a case report with necropsy finding *Ann Int Med* 43 1107 1955
- 629 HECHT H H AND LANGER R I Personal communication
- 630 HECHT H H AND MYERS C B Auricular heart sounds in auricular flutter *Am Heart J* 23 610 1945
- 631 HEERLIN R Ueber die sog energetischen dynamischen Herztunfirien Klin Wochenschr 27 330 1949
- 632 HEITMANN M R BRADFORD J Y AND MILLER J V Myocarditis and Friedrich's ataxia report of 2 cases *Am Heart J* 38 757 1919
- 633 HEILFELDER I Durozier et son oeuvre Contribution a l'histoire de la cardiologie Paris thesis 4430 1922
- 634 HELWIG C A Atheromatosis of the mitral valve *Am Heart J* 24 41 1942
- 635 HELLS I P W AND McDONALD D A Arterial blood flow calculated from pressure gradient *J Physiol* 124 30 1954
- 636 HELLS I P W AND McDONALD D A Observations on luminal flow in veins *J Physiol* 124 611 1944
- 637 HELLS I P W AND McDONALD D A Streamline flow in vein *J Physiol* 126 5 1944
- 638 HELLS I P W AND McDONALD D A Systolic backflow in the dog femoral artery *J Physiol* 122 73 1953
- 639 HELWIG F C The frequency of anomalous reticuli in the right atrium of the human heart—Chamber network report of a case *Am J Path* 8 71 1912
- 640 HENDERSON Y AND JOHNSON J I Two modes of closure of the heart valve *Heart* 4 69 1912
- 641 HENN M J PARKIN T W HANCAVERES M M AND OBER N M Acute systemic lupus erythematosus syndrome from hydralazine hydrochloride *Arch Int Med* 95 857 1950
- 642 HENON H Beitr z Kinderh p 170 1951
- 643 HENRY J N Report of a case of loud venous hum heard over the xiphoid cartilage in curvature of the liver autopsy *Am J Med Sci* 143 72 1911
- 644 HEPPER N C C BURCHELL H B AND EDWARDS J I Cardiac Clinic CIVI Mitral insufficiency in healed unrecognized bacterial endocarditis Proc Staff Meet Mayo Clin 31 609 1956
- 645 HERRICK I C An experimental study into the cause of the increased portal pressure in portal cirrhosis *J Exper Med* 9 93 1907
- 646 HERRICK J B A Short History of Cardiology Charles C Thomas Springfield 1947

- 60 HERRMANN C R AND SCHOFIELD A D The incidence of rupture of the aortic root or sinus of Valvula aortae into the right atrium Am Heart J 15 1931
- 61 HERRMAN E F AND LEO C F Note on the sound accompanying the single contraction of skeletal muscle J Physiol 8 2 1935
- 62 HERON R V AND SPENCE M Ruptured congenital aneurysm of posterior sinus of Valvula aortae Heart J 8 1936
- 63 HE W R Über die periphere Registrierung der Blutströmung Pflüger Arch exp Physiol 128 379 1931
- 64 HICKMAN J B Atrial septal defect a study of intracardiac shunt ventricular output and pulmonary pressure gradient Am Heart J 38 501 1949
- 65 HILLIARD R F AND MORRIS R C Note on the transmission of noise by the mammae to the abdominal aorta Am Heart J 7 1937
- 66 HILLYER T F AND COLEBY R Congenital mitral stenosis Bornigen study of its manifestations Am J Roentgenol 76 73 1942
- 67 HILLIARD J K AND FISKE W T Condenser microphones for measurement of high sound pressures Altec Lansing Corporation 93-5 Santa Monica Blvd Beverly Hills Calif 1936 Mimeograph 1
- 68 HILLIARD J K AND NOBLE J J The lip-sh cone as microphone system for the JEF Electrocardiogram on Audio Vol 11 2 No 11 1934
- 69 HINOHARA S Cases of thromboangitis obliterans in which a venous valve murmur was recorded through the esophagus Am Heart J 2 36 1943
- 70 HINOHARA S Systolic gallop rhythm Am Heart J 22 1931
- 71 HIPPOCRATE De morbis 6 an 15 b ii 4<sup>a</sup> ni 18 d De intern aff Sec 23 c Irenor 474 f De Le in Homine Sec 14 p Irogen Sec 14 h De morbis ii Sec 61 i ii Sec 39 j iii Sec j
- 72 HIPPOCRATE Prognostic signs Jones translation 2<sup>a</sup> Harvard Univ Cambridge Mass 1937
- 73 HIRSH E AND BARRELL G Muehlenberg's als symptom einer Lymphstase des rechten Herzens beim künstlichen Pneumothorax Wien klin Wchn tsr 48 23<sup>r</sup> 1935
- 74 HIRSCHFELD A D Diseases of the Heart and Lungs J B Lippincott Co Philadelphia 1930
- 75 HIRSCHFELD A D Some variations in the form of the venous pulse Preliminary report Bull John Hopkins Hosp 18 265 190
- 76 HOFFER R Ein Verfahren zur Demonstration der Aktionsstrom-Nachtrag Arch exp Physiol 977 305 1919
- 77 HUBBARD M D Mechanism of Semilunar flaps of the heart Zapisch ein Beitrag zur Frage eines völlig verflochten Schließens derselben Deut ches Arch klin Med 164 131 1927
- 78 HUNTER T On the structure of the valves of the heart London 1733 433 1738 n
- 79 HUNTER J Treatise on Diseases of the Heart 1743
- 80 HURRY M S AND BLUNT C Jr Murmurs of a talched rheumatic valvular disease Bull of Denver Rheumatic Fever Diagnostic Service May 1930
- 81 HURRY H Über ophagische Kulturation Centralblatt für klin Med No 44 Oct 16 1932
- 82 HURRY H Ein Fall von Mitralinsuffizienz festgestellt durch einen Tumor am linken Vorhof Zeitschrift für Herz und Gefäßkrankheit 4 9 1912
- 83 HURRY H M LUTHER I W AND MEHLER R The phonocardiogram in spontaneous interstitial emphysema of the mediastinum Am Heart J 20 1937
- 84 HOLLOACK H Die Bedeutung der Sinusphysiologie des Herzes für die tägliche Praxis der Herz-kulturation Deut che med Wchn chr 74 119 1919
- 85 HOLLOACK H Die Phonokardiographie Regeln von Med u Kinderhe 3 46<sup>r</sup> 1937
- 86 HOLLOACK H CALVERT W AND STERNBERG J Distal chest auscultation in ductus arteriosus Cardiologia 31 170 1937
- 87 HOLLOACK H AND KATZ J Chest girth circumference and peripheral circulation Deut che med Wchn chr 78 41 1933
- 88 HOLLOACK H AND LAUREN B Die diagnostische Bedeutung der endformalen distalen Brustgirthmessung Arch klin Med 199 151 1937
- 89 HOLLOACK H AND WOLF D Atla und kurgelsches Lehrbuch der Phonokardiographie 2<sup>e</sup> ed Theme Stuttgart 1931
- 90 HOLLOACK H The anatomical appearance of rheumatic tricuspid valve disease Brit Heart J 19 111 1937
- 91 HOLLOACK H W The stethoscope and its use Bull p 531 Willam and Rev (ref 1936)
- 92 HOLY E Gallop rhythm Am Heart J 2 451 1937
- 93 HOOPER R The Posthumous Works of Robert Hooper containing his Coleridge Lectures and Other Discourses Read at the Meeting of the Illustrations Royal Society etc Published by Sam Smith and Benj Walford (Printed by the Royal Society) at the Index Arms in St Paul Churchyard 1760 1 39 (The method of improving natural philosophy)
- 94 HOPE J A Treatise on the Diseases of the Heart

- and Great Vessels etc London 1839 (3rd edition) (a P 118 b P 470 c P 391 d I 87)
- 712 HORNICK C B Ueber das sogenante Mullen geräusch München med Wehnschr 69 819 1922
- 713 HOUSSAY B A Les bruits du coeur Presse méd 11 1353 1936
- 714 HOWARD C P Aortic insufficiency due to rupture by strain of a normal aortic valve Canad M A J 10 12 1926
- 715 HUCHARD H Contribution à l'étude clinique des tendons aberrants du coeur Rev de méd 13 113 1893
- 716 HUCHARD H Exemple de souffles cardio pulmonaires à foyers multiples très intenses simulants un rétrécissement mitral avec insuffisance et surtout une insuffisance aortique Bull Soc méd Hop Paris 3 288 1896
- 717 HUCHARD H Les maladies du coeur et leur traitement J B Baillière Paris 1908
- 718 HUCHARD H Traité des maladies du coeur Vol 32 p 640 1906
- 719 HUDSON R The normal and abnormal interatrial septum Brit Heart J 17 489 1959
- 720 HUFTHILE K Ueber die mechanische Registrierung der Herztonen I Flugz Arch ges Physiol 60 263 1905
- 721 HUFTHILE K Ueber eine Methode zur mechanischen Registrierung der Töne Deutsche med Wehnschr 19 77 1903
- 722 HUGGINS W H A phase principle for complex frequency analysis and its application in auditory theory J Acoust Soc Am 24 582 1952
- 723 HULL I Cause and effects of flow through defects of atrial septum Am Heart J 38 350 1949
- 724 HULTGREN H N Calcific disease of the aortic valve Arch Path 45 694 1945
- 725 HULTGREN H N Origin of the early systolic click of the pulmonary artery Stanford M Bul 14 183 1956
- 725A HULTGREN H N Intensity of the first heart sound in mitral stenosis with atrial fibrillation Clin Res 6 84 1958
- 725B HULTGREN H AND GALL E Ostent deformans and calcific disease of the heart valve Am Fed for Clin Research Atlantic City May 4 1958
- 726 HULTGREN H N AND LEO T I First heart sound in mitral stenosis Abstract Circulation 16 895 1957
- 727 HULTGREN H N SEIZER A LURDY A HOLMAN I AND GREGORY F The syndrome of patent ductus arteriosus with pulmonary hypertension Circulation 15 1953
- 728 HULTING B AND VEDSALL A Coarctation of the aorta in unusual site Acta radiol 43 453 1955
- 729 HUNT F V *Electroacoustics The Analysis of Transduction and Its Historical Background* John Wiley & Sons New York 1954
- 730 HUNTER A The heart in anemia Quart J Med 15 107 1946
- 731 HUNTER W *Medical Observations and Researches* Vol II p 403 1764
- 732 HURLBETT F JR Perikardies A treatise on the heart from the Hippocratic corpus Introduction and translation Bull Hist Med 7 1104 1939
- 733 HURST J W Personal communication
- 734 HURST J W Some comments on cultivation of the heart I The intensity of the first heart sound at the apex J M A Georgia 46 17 1957
- 735 HURST W W AND SCHENK F R High ventricular septal defect with slight dextroposition of the aorta (Liemenger type) which prevented the clinical features of patent ductus arteriosus Am Heart J 36 144 1948
- 736 HUXLEY L *Life and Letters of Thomas Henry Huxley* Vol 1 p 439 D Appleton & Co Inc New York 1901
- 737 IANSON T W AND POLLACK B I Coarctation of the abdominal aorta Am Heart J 11 314 1956
- 738 IRONS I E AND JENNINGS A F Presystolic murmur in rapid hearts simulating murmur of mitral stenosis with report of necropsies J A M A 78 957 1922
- 739A IRVING R I Jame Hope and the history of aortic regurgitation Can J Ho p Rep 106 1 1957
- 739 IRVING R L Outcome of uncomplicated aortic mitral Brit M J 1 432 1956
- 739A ISAACS J P BERGLAND E AND SARNOFF S J Ventricular function III The pathologic physiology of acute cardiac tamponade studied by means of ventricular function curves Am Heart J 48 66 1954
- 740 JACKSON F Constrictive pericarditis and mitral stenosis Proc Roy Soc Med 11 311 1950
- 741 JACOBSON G COSBY R S CRIFFITH G C AND MEYER B V Avascular stenosis as cause of death in surgically treated coarctation of aorta Am Heart J 45 889 1953
- 742 JACOB B V Noninfectious thrombosis of a patent ductus arteriosus Am Heart J 20 236 1910
- 743 JAMES W B Pneumopericardium Tr A Am Physicians 11 351 1904
- 744 JEANS SIR JAMES *Science and Music* Macmillan New York 1937
- 745 JERRARD W AND BURTON A C Demonstration of hemodynamic principle in particular of turbulence and stream lined flow J Appl Physiol 4 620 1952
- 746 JERVEILL A Induced disease Am Heart J 47 780 1954
- 747 JOACHIM C AND WEISS O Registrierungen von Herztonen und Herzgeräuschen beim Menschen Deutsches Arch Klin Med 98 513 1910
- 748 JOHANSSON B The electrocardiogram and phono

- radiogram of the non hibernating hedgehog  
Cardiologia 30 3: 136
- 11 JONES T N P WILLIAMS C R AND BLACKLOCK A The anatomy of pulmonary stenosis and atresia with comment on surgical therapy Surgery 116: 1953
- 12 JOHNSON J B JORDAN A AND LAWLAN J W Arteriovenous fistula simulating patent ductus arteriosus Evaluation by venous catheterization and angiocardiology J A M A 165 146: 1951
- 13 JONES F D Extra sound occurring in ear disease tale Am Heart J 15 221 1975
- 14 JOHNSON F D Symposium on cardiovascular sound Circulation 18 70-76 411-437 1957
- 15 JONES F D AND KLINE F An acoustic study of the telescope Arch Int Med 55 328 1940
- 16 JONES F D AND OSERY D C Vibration of the femoral artery over the precordium Circulation 3 59 1951
- 17 JONES A Heart Disease in Infancy 2 J B Harvey and Blissett London 1951
- 18 JONES A M AND LANCELEY J A Aortic sinus aneurysm Brit Heart J 11 355 1949
- 19 JONES F C AND RANDOLPH H Congenital complete heart block diagnosed in utero with and tracings and multilevel electrocardiogram of the mother Am Heart J 23 109 1947
- 20 JOSEPH C Geschichte der Physiologie der Herzzeit Janu 2 193 315-373 50-57 1947
- 21 JOSEPH C AND BERNIERE C Histoclement mit der Frequenz eines Ruffes pro taktische malgularhythmie complete Bull et mfm soc med Hop Lar p 516 March 21 1950
- 22 JONES J D FERRY M M AND SLOAN H F Jr Left atrial electrocardiography in mitral insufficiency in man a correlative study by angiocardiology left heart catheterization and experimental production in dog (Abstract) Circulation 18 895 1957
- 23 JONES D AND DAVIE L C Heart sound and intracardiac pressure in mitral stenosis Brit Heart J 10 48 1952
- 24 JERUSALEM A J Anomalous left coronary artery Am Heart J 11 429 1952
- 25 KAHN R H Du Herle I H K Hugers Arch ges Physiol 164 1 1913
- 26 KAHN R H Studien am Fh n Laryngogramm Hugers Arch ges Physiol 140 4 1 1911
- 27 KALMAN A J SCHNEPPEL J B AND STRAUS B Chronic constrictive pericarditis and rheumatic heart disease Am Heart J 15 201 1953
- 28 KANYANIAN A D AND DE JONG R N Familial primary amyloidosis with nervous system involvement Neurology 3 399 1953
- 29 KANTWITZ A KANTWITZ S AND HERMANOWITZ A A cinematographic study of the function of the mitral valve in situ In Surgical Forum—Clinical Congress of the American College of Surgeons p 701 W B Saunders Co Philadelphia 1951
- 30 KATZ H T AND KOLLESKY S Calcific Disease of the Aortic Valve Lippincott Philadelphia 1947
- 31 KATZMAN A M De l'origine de l'insuffisance du coeur Arch mal coeur 29 75 1958
- 32 KATZ L N The mechanism of right and left ventricular contraction and the independent variation in their duration Am J Physiol 72 3 1952
- 33 KATZMAN S I AND MURKHAM J W Correlation of the abdominal aorta with death from rupture of an aneurysm of a cerebral artery Ann Int Med 11 418 1950
- 34 KATZ C F WILSON J W JONES H F AND BRADSHAW J M The validity of the electromagnetic method for measurement of diameter change of the aorta and pulmonary artery during circulatory disturbance J Clin Invest 28 225 1949
- 35 KATZ J H AND THURMAN W Experimental production of pulmonary insufficiency Arch Surg 59 716 1954
- 36 KATZ H D Le cœur du Vainqueur Mémento of the Heart and Blood Lippincott Philadelphia 1950
- 37 KATZ J H Variation in the first curve of the heart and the AV conduction time in rheumatic fever of children Arch Dis Child 32 21 1957
- 38 KATZ J D AND FORTNER C Aortography in infant Circulation 2 40 1950
- 39 KATZ J D ROWE R D AND VLADEY AND HANSEN J H Complete anomalous pulmonary venous drainage Am J Med 10 23 1951
- 40 KATZ J D ROWE R D AND VLADEY I Heart Disease in Infancy and Childhood Macmillan Co New York 1958
- 41 KATZ J J Jr Diagnostic value of phonocardiography in mitral stenosis Mode of production of first heart sound Am J Med 19 66 1955
- 42 KELLY J J Jr Symposium on cardiovascular Circulation 18 70 1957
- 43 KERR A JR Schaefer Bacterial Endocarditis Charles C Thomas Springfield 1950
- 44 KERR A JR AND CO C M Retention of embolic relationship of aortic and pulmonary valve cusps and a suggested nomenclature Anat Rec 125 77 1956
- 45 KERR A JR AND FALMER F D The Balthazar-Foster murmur and Foster's rule New England J Med 248 1064 1953
- 46 KERR W J Clinical diagnosis of congenital heart murmurs by means of the sphygmophone J Pediatr 37 569 1951
- 47 KERR W J Stethoscope and sphygmophone In



- Medical Physics Year Book Publishers Inc Chicago 1944*
- 785 KERR W J The symbolophone a double stethoscope for the comparison and lateralization of sound In *Essays in Biology* University of California Press Berkeley, 1943
  - 786 KERR W J ALTHAUSSEN T I BASSETT A M AND GOLDMAN M J The symbolophone a modified stethoscope for the lateralization and comparison of sound *Am Heart J* 14 594 1937
  - 787 KERR W J AND HARI V C JR Transmission of murmurs *Am Heart J* 37 100 1949
  - 788 KERR W J HARI V C RAJAPOOTI I AND BIERMAN H R The propagation of murmurs and the local production of valvular murmurs in relation to the pulse waves *Tr A Am Physicians* 61 308 1948
  - 789 KEYNES G *The Portraiture of William Harvey* Royal College of Surgeons of England London 1949
  - 790 KEYS A AND SHAIRO M I Patency of the ductus arteriosus *Am Heart J* 66 153 1943
  - 791 KIDDER G J MURPHY C F AND WAGGONER J N Rupture of papillary heart muscle following myocardial infarction with present survival of over three years *Ill M J* 110 28 1956
  - 792 KILBY R A DUSHANE J W WOOD L H AND BURCHFIELD H B Bicuspid malformation a clinical and laboratory study *Medicine* 35 161 1956
  - 793 KILLIP T III AND LUKAS D H Tricuspid stenosis Physiologic criteria for diagnosis and hemodynamic abnormalities *Circulation* 16 3 1957
  - 794 KILOH G A Pure aortic stenosis *Brit Heart J* 12 33 1950
  - 795 KING J T The clinical recognition and physical signs of bundle branch block *Am Heart J* 3 505 1925
  - 796 KING J T AND MELACHERN D The nature of the physical signs of bundle branch block *Am J M Sc* 183 445 1932
  - 797 KING T W An essay on the safety valve function in the right ventricle of the human heart and the gradations of this function in the circulation of warm blooded animals *Cuy's Hosp Rep* 2 104 1837
  - 798 KISCH B Heart sounds in tachycardia *Tr Am Coll Cardiol* 2 232 1952
  - 799 KISSANE R W KOONS R A AND CLARK T E Traumatic rupture of the aortic valve *Am J Med* 4 606 1948
  - 800 KISSIN M Pulmonary insufficiency with a super numerical cusp in the pulmonary valve Report of a case with review of the literature *Am Heart J* 12 206 1936
  - 801 KIWISH VON ROTTERAU F A Neue Forschungen über die Schallerzeugung in den Kreislauforganen Verhandl d Physikalisch Medicinischen Gesellschaft in Würzburg 1 6 1850
  - 802 KJELBERG S R RIDGHE V AND SJÖSTRAND T The effect of adrenaline on the contraction of the human heart under normal circulatory condition *Acta physiol Scandinav* 24 333 1952
  - 803 KJELBERG S R RIDGHE V AND SJÖSTRAND T Influence of autonomic nervous system on contraction of human heart *Acta physiol Scandinav* 24 350 1952
  - 804 KLASSEN K P AND MEYERSTROTH C V Cinematographic demonstration of valvular disorders *Surgical Forum* 5 52 1954
  - 805 KLEIN J B LEATHAM A IAN C MAAS H AND MINOT G Standardization of phonocardiography *Cardiology* 26 262 1955
  - 806 KLINEFELTER H F Heart in sickle cell anemia *Am J M Sc* 203 34 1947
  - 807 KNORRICH R AND RAMSON A J Arteriovenous fistula of the heart *Am Heart J* 50 474 1956
  - 808 KNOWLES J H CORBIN R AND STOREY C F Clinical test for pulmonary congestion with use of the Valalva maneuver *J A M A* 160 44 1956
  - 809 KOLETSKY S Bicuspid aortic valve and bacterial endocarditis *Am Heart J* 26 343 1943
  - 810 KOLETSKY S Congenital bicuspid aortic valve *Arch Int Med* 67 129 1941
  - 811 KOLETSKY S Congenital bicuspid pulmonary valve *Arch Path* 31 339 1941
  - 812 KOVAY R CHAUDHURY D C R AND BASU A K Aortic septal defect *J Indian M A* 27 61 1956
  - 813 KOVAY R CHAUDHURY D C R AND BASU A K A case of coarctation of aorta at an unusual site *Am Heart J* 49 275 1955
  - 814 KORNER P AND SMITHINGFORD J Tricuspid incompetence and right ventricular output in congestive heart failure *Brit Heart J* 19 1 1957
  - 815 KORNYA H M The nature and time relation of the compression sounds of Korotkoff in man (a study of 50 patients) *Am J Physiol* 76 247 1926
  - 816 KOROTKOFF N S A contribution to the problem of method for the determination of the blood pressure *Rep Imper Milit Med Acad St Petersburg* 11 365 1905 For translation see Lewis (1906) and Pickering (1908)
  - 817 KOSMANN C L The opening snap of the tricuspid valve a physical sign of tricuspid stenosis *Circulation* 11 378 1955
  - 818 KOLNITZ W B CILSON A S AND SMITH J R The use of the cathode ray for recording heart sounds and vibrations II Studie on the normal heart *Am Heart J* 20 667 1940

- 61 KATZ W B AND SMITH J R Studies on the early recognition of myocardial disease by use of the vibrocardiogram South M J 35 413 194
- 62 KATZ W B SMITH J R AND WRIGHT S T Observation on the effect of tourniquet on acute cardiac arrest normal ulcers in chronic heart failure Am Heart J 21 694 1941
- 63 KATZ W B AND WRIGHT S T Comparison of total vibration obtained from a normal rapidly dying human heart with that obtained in chronic myocardial disease Am Heart J 27 896 1944
- 64 KATZ L A LARER R I AND CORBIN A J Hemodynamics of patent ductus arteriosus analysis by simultaneous simultaneous pressure recording during uterine J Appl Physiol 11 161 1947
- 65 KATZ V E AND HILLBICK K Untersuchungen am Herzen bei akutem Herzversagen Verh Anat 26 334 1940
- 66 KATZ K Untersuchungen über die mechanische und elektrische Leistung des Herzes zum Totalen der energetischen dynamischen Herzen affizienz (Hegghip) Cardiologia 23 300 1943
- 67 KATZ K AND IEREN I Myxoma of heart polypoid tumor of left atrium diagnosed ante mortem Acta med Scandinav 143 340 1947
- 68 KATZ T The gallop sound Studies of the mechanism of production Circulation 18 276 1940
- 69 KATZ T HILBERT J A AND KATZ T Th mechanism of gallop sound (study) with the aid of the electrokymograph Ann Int Med 11 136 1941
- 70 KATZ T AND SCHWABEL T C Jr Certain abnormal circulatory dynamics of mitral stenosis associated with a major degree of regurgitation Am J Med 18 10 1943
- 71 KATZ T SCHWABEL T C JR BLAKEMORE W S AND WHEAT A F Diastolic gallop sound the mechanism of production J Clin Invest 26 1030 1947
- 72 KLEINER C Die ersten Klappen des Herzens und ihre Verengung n Fronten Neurol 316 1940
- 73 KLEINER A C AND MARKOWITZ M The diagnosis of mitral insufficiency in rheumatic children Am Heart J 25 718 1949
- 74 LAENNE R T H The Heart and its Diseases 11th ed 1914 1919 1924 1929 1934 1939 1944 1949 1954 1959 1964 1969 1974 1979 1984 1989 1994 1999 2004 2009 2014 2019 2024 2029 2034 2039 2044 2049 2054 2059 2064 2069 2074 2079 2084 2089 2094 2099 2104 2109 2114 2119 2124 2129 2134 2139 2144 2149 2154 2159 2164 2169 2174 2179 2184 2189 2194 2199 2204 2209 2214 2219 2224 2229 2234 2239 2244 2249 2254 2259 2264 2269 2274 2279 2284 2289 2294 2299 2304 2309 2314 2319 2324 2329 2334 2339 2344 2349 2354 2359 2364 2369 2374 2379 2384 2389 2394 2399 2404 2409 2414 2419 2424 2429 2434 2439 2444 2449 2454 2459 2464 2469 2474 2479 2484 2489 2494 2499 2504 2509 2514 2519 2524 2529 2534 2539 2544 2549 2554 2559 2564 2569 2574 2579 2584 2589 2594 2599 2604 2609 2614 2619 2624 2629 2634 2639 2644 2649 2654 2659 2664 2669 2674 2679 2684 2689 2694 2699 2704 2709 2714 2719 2724 2729 2734 2739 2744 2749 2754 2759 2764 2769 2774 2779 2784 2789 2794 2799 2804 2809 2814 2819 2824 2829 2834 2839 2844 2849 2854 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- phonocardiographique) Arch mal coeur 29 665 1936
- 851 LAUBRY C ROLIER D AND VAN BOGAERT A D Deux signes cliniques nouveaux de la tachycardie auriculaire (auricular flutter) Arch mal coeur 25 321 1932
- 852 LAUBRY C AND THIROLOUX P I Sur la pathologie et la valeur clinique des souffles organiques du coeur Pre se méd 40 1109 1932
- 853 LAUSON H D BLOOMFIELD R A AND COURTNEY A The influence of the respiration on the circulation of man with special reference to pressures in the right auricle right ventricle femoral artery and peripheral veins Am J Med 1 315 1946
- 854 LAVELLE F ENDERIE J AND MEERSSEMAN F Communication interventriculaire avec anomalies des valvules aortiques à la fois d'origine congénitale et acquise Acta cardiol 11 289 1956
- 855 LAVELLE F TYBERCHEIN J BRASSEUR J AND MEERSSEMAN F Complexe d'insuffisance avec insuffisance pulmonaire par abaissement des valvules Acta cardiol 9 249 1954
- 856 LEATHAM A A classification of systolic murmurs Brit Heart J 17 574 1955
- 857 LEATHAM A Peroral communication
- 858 LEATHAM A The phonocardiogram of aortic stenosis Brit Heart J 13 153 1951
- 859 LEATHAM A Phonocardiography Brit Med Bull 8 334 1952
- 860 LEATHAM A Rheumatic aortic incompetence with delayed diastolic murmur on auscultation Proc Roy Soc Med 43 309 1950
- 861 LEATHAM A Splitting of the first and second heart sounds J med 2 607 1954
- 862 LEATHAM A Symposium on cardiovascular sound Circulation 16 414-436 1957
- 862A LEATHAM A Systolic murmurs Circulation 17 601 1958
- 863 LEATHAM A AND CRAWFORD I Auscultatory and phonocardiographic signs of mitral septal defect Brit Heart J 11 193 1956
- 864 LEATHAM A AND TOWERS M Splitting of the second heart sound in health Brit Heart J 13 575 1951
- 865 LEATHAM A AND VOCHTEL I The early systolic sound in dilatation of the pulmonary artery Brit Heart J 16 21 1954
- 866 LEATHAM A AND WEITZMAN D W Auscultatory and phonocardiographic signs of pulmonary stenosis Brit Heart J 19 303 1957
- 867 LEE G DE J AND LAMETTE T M D A simple test for interatrial communication Brit Med J 1 1278 1957
- 868 LEFLOU N M BRUN F S AND MANNING C W An opening snap recorded in a case of tumor of the left atrium New England J Med 257 178 1957
- 869 LECOUST I Limousin Ode Mort 1630 Cited by Gunn and Wood (621)
- 870 LEIGHT I SNIDER T H CLIFFORD C O AND HELLEMS H K Hemodynamic studies in sickle cell anemia Circulation 10 633 1954
- 871 LEIARD M C The stethoscope Some note on its history J Nat M A 47 177 1955
- 872 LEJEUNE DE KERGADEZ J A Mémoire sur l'auscultation appliquée à l'étude du gros et du petit cœur ou Recherche sur deux nouveaux signes propres à faire reconnaître plusieurs circonstances de l'état de gestation J de physiol expér 2 117 1822
- 873 LEONARD J J HARVEY W I AND HUNAGEL C A Rupture of the aortic valve New England J Med 252 205 1955
- 874 LEONARD J J AND MOSER K M Calcification of the ascending aorta A study of its diagnostic specificity for aortic aortitis Abstract Circulation 16 905 1957
- 875 LEONARD J J WEISSELER A M AND WARREN J V Observations on the significance of the delayed appearance of the first heart sound in mitral stenosis Abstract Circulation 16 906 1957
- 876 LEONARD J J WEISSELER A M AND WARREN J V Observation on the mechanism of the aortic gallop rhythm Circulation 17 1005 1958
- 877 LIEBERMAN F On the relation between the site of valvular involvement in endocarditis and the blood pressure resting on the valve Am J Med 24 318 1952
- 878 LIEBERMAN F A quantitative stethoscope and its clinical application Am Heart J 43 581 1952
- 879 LIEBERMAN E Symposium on cardiovascular sound Circulation 16 270-290 414-436 1957
- 880 LIEBERMAN L AND SWARICZ B The measurement of the Q-T interval of the electrocardiogram Circulation 6 375 1952
- 881 LIEBERMAN J AND MEANS J H Cardiovascular symptomatology in exophthalmic goiter Am Heart J 8 55 1932
- 882 LEROY C V AND ROBERTS R C Systolic gallop rhythm as a sign of necrosis of the left ventricle Am Heart J 21 115 1941
- 883 LEBESON M AND BRIDEN W Systolic murmurs in healthy children and in children with rheumatic fever J med 2 673 1957
- 884 LETHIER M Anatomie du couvent (étendu à la totalité de l'orte et pontamment guerrière d'un effluve aortique avec intégrité parfaite de valvule aigmoïdes Bull et mém Soc méd hôp de Paris 22 1045 1905
- 885 LEVINE S A Clinical Heart Disease 1 ed 3 p 69 W B Saunders Co Philadelphia 1945
- 886 LEVINE S A The clinical recognition of paroxysmal ventricular tachycardia Am Heart J 3 177 1927-1928

- 67 LEVINE S A Diagnostic value of cardiac auscultation *J A M A* 141 509 1949
- 68 LEVINE S A AND HARVEY W I *Clinical Investigation of the Heart* W B Saunders Co Philadelphia 1949
- 69 LEVINE S A AND LIAOFF W B Some note on the transmission of heart murmurs *Ann Int Med* 21 99 1944
- 70 LEVINE S A AND LOVE D I Mitral stenosis without murmurs *Circulation* 21 599 1950
- 71 LEVIN D C LEMADES D T AND CRIFFITH G C Detecting aneurysm of the aorta by clinical electrocardiographic and laboratory features: A report of 5 autopsies *Circulation* 1 330 1950
- 72 LEVINSON D C MEEHAN J I JR SCHWARTZ L H AND CRIFFITH C C Rapid registration of cardiac thrill in acquired and congenital heart disease *Circulation* 24 479 1956
- 73 LEWIS D D W Congenital arteriovenous fistulae *Lancet* 2 691 1950
- 74 LEWIS D H Experiences with the Jerrard Burton flow demonstration in the teaching of hemodynamics *J Appl Physiol* 6 716 1953
- 75 LEWIS D H DEITZ C W WALLACE J D AND BROWN J H JR Studies in intracardiac phonocardiography *Abstract Circulation* 24 96 1956
- 76 LEWIS D H DEITZ C W WALLACE J D AND BROWN J R Intracardiac phonocardiography in man *Circulation* 18 64 1957
- 77 LEWIS J K Nature and significance of heart sound and of apex impulse in bundle branch block *Arch Int Med* 63 743 1953
- 78 LEWIS J K AND DEWEY W The origin of heart sound and their variation in myocardial infarction *J A M A* 115 771 1935
- 79 LEWIS J K AND HEWLETT A W The cause of increased vascular sound after epinephrine injection *Heart* 10 1 1923
- 80 LEWIS T Congenital tricuspid stenosis *Clin Sci* 5 261 1946
- 81 LEWIS T Disease of the Heart p 131 Macmillan New York 1933
- 82 LEWIS T Illustrations of heart sound record *Quart J Med* 6 441 1913
- 83 LEWIS T The relation of auricular systole to heart sound and murmurs *In Lectures on the Heart* p 53 Paul H Hoeber New York 1915
- 84 LEWIS T The time relation of heart sound and murmurs with special reference to the acoustic sign of mitral stenosis *Heart* 4 241 1913
- 85 LEWIS T AND STOKES J A Curious syndrome with suggestive cervical arteriovenous fistula with pulse of neck and arm lost *Brit Heart J* 4 5 1947
- 86 LEWIS W H JR The evolution of clinical sphygmomanometry *Bull New York Acad Med* 17 871 1941
- 87 LIEBEN I von Ueber die Sclerose der Coronar Arterien und die davon abhängigen Krankheiten zu finden *Ztschr klin Med* 7 459 and 539 1884
- 88 LIAN C Absence de vibration auriculaires dans la constitution du premier bruit du cœur *Compt Rend Soc Biol* 135 791 1942
- 89 LIAN C Du diagnostic des souffles systoliques aigus et de l'insuffisance mitrale fonctionnelle *Lancet* the 14 1939
- 90 LIAN C Etude expérimentale de l'insuffisance mitrale fonctionnelle *Arch mal cœur* 2 1 1909
- 91 LIAN C L'auscultation cardio-océphalienne *Arch mal cœur* 38 271 1941
- 92 LIAN C L'auscultation du creux du sternum *Arch mal cœur* 3 48 1918
- 93 LIAN C L'auscultation du sternum Son importance égale dans le diagnostic du rétrécissement mitral *Lancet* 149 1363 1941
- 94 LIAN C Les vibrations cardiaques systoliques anasthétiques de la calcification du péricarde *Bull et mém Soc méd hôp Paris* 3 1941
- 95 LIAN C Le diagnostic stéthoscopique de la calcification du péricarde grâce à la vibration péricardique systolique *Bull et mém Soc méd hôp Paris* p 97 June 23 1944
- 96 LIAN C Le souffle veineux continu de la base inter-épineuro-vertébrale droit *Bull et mém Soc méd hôp Paris* 53 1100 1937
- 97 LIAN C The use of the phonocardiograph in clinical cardiology *Brit Heart J* 10 97 1948
- 98 LIAN C AND BRACON C Importance et interprétation de l'influence du rapport entre les contractions auriculaires et ventriculaires sur l'intensité du premier bruit dans la dysfonction auriculo-ventriculaire (bruit de canon) et dans le rythme sinu-aural *Arch mal cœur* 41 360 1948
- 99 LIAN C CHEVALIER H CORLESTE B AND DUBREUIL H Le déroulement du deuxième bruit dans les types de branchement *Arch mal cœur* 42 513 1949
- 100 LIAN C AND DEPARIS M Le claquement mitral systolique pleuro-péricardique *Bull et mém Soc méd hôp Paris* 49 1933
- 101 LIAN C AND CLEMENS P Importance du signe de la zone silencieuse supramittale dans le diagnostic du rétrécissement aortique pur ou associé à l'insuffisance aortique *Lancet* 1 58 1141 1939
- 102 LIAN C GILBERT DREYFUS AND FLECH P Importance de la recherche et de la constatation d'un souffle systolique du creux du claviculaire pour le diagnostic des artérites incomplètement oblitérantes de la sous-clavière *Bull et mém Soc méd hôp Paris* 1901 July 29 1907
- 103 LIAN C AND CORLESTE A Les bruits du cœur

- fœtal in utero (étude phonocardiographique)  
Arch mal coeur 31 173 1938
- 924 LIAN C AND GOLBLIN V Les bruits du cœur  
fœtal in utero (étude phonocardiographique)  
Bull Soc Gynec et Obst 2 423 1938
- 925 LIAN C GOLBLIN V AND BARAIGE E Diag-  
nostic et valeur étiologique de l'allongement  
et du raccourcissement de la systole ventricu-  
laire la constante systolo diastolique Pre e  
méd 42 787 Arch mal coeur 33 241 1939
- 926 LIAN C AND CLINARD P L'auscultation dans le  
premier espace intercostal droit Son importance  
pratique Arch mal coeur 39 92 1946
- 927 LIAN C AND HUBERT L Intérêt de l'épreuve  
d'effort dans le diagnostic du bruit de galop  
Arch mal coeur 41 175 1948
- 928 LIAN C AND HUBERT E L'épreuve d'effort dans  
l'étude du premier bruit en dehors de l'insuf-  
fisance cardiaque Arch mal coeur 41 181 1948
- 929 LIAN C LYON CAEN I AND DUMÉRY R Le  
souffle continu thyroïdien supérieur signe  
caractéristique du syndrome bradonien Pre e  
méd 41 1012 1933
- 930 LIAN C MARCHAL M AND PAUTRAT J Un  
signe clinique de la calcification du péricarde  
la vibration péricardique protodiastolique Bull  
et mém Soc méd hôp Paris p 20 January 13  
1933
- 931 LIAN C MINOT C HEBERT V AND RACER V  
Relations chronologiques entre les phénomènes  
mécaniques et électriques du cœur Arch mal  
coeur 48 39 1933
- 932 LIAN C MINOT C AND WELTI J J Phono-  
cardiographie Auscultation collective Ma on et  
Cie Paris 1941
- 933 LIAN C AND OBISET J De l'existence d'un  
double bruit par la percussion abdominale dans  
l'ascite et de son importance diagnostique Bull  
et mém Soc méd hôp Paris 47 1402 1931  
Pre e méd 42 1337 1934
- 934 LIAN C AND RACINE M La phonocardiographie  
I Technique et résultats normaux Ann méd  
p 69 June 1933
- 935 LIAN C AND RACINE M La phonocardiographie  
II Affections valvulaires et congénitales du  
cœur Ann méd p 75 June 1933
- 936 LIAN C AND RACINE M La phonocardiographie  
III Les rythmes à trois temps Ann méd p  
157 July 1933
- 937 LIAN C AND TURSZ M Le diagnostic clinique  
entre le galop pré-systolique retardé et le dc-  
doublement du premier bruit Arch mal coeur  
42 607 1949
- 938 LIAN C AND VILENSKI J Fréquence du troisième  
bruit du cœur dans l'insuffisance mitrale pure  
Cause d'erreur dans le diagnostic de maladie  
mitrale Pre e méd 52 503 1954
- 939 LIAN C AND WELTI J J Le claquement artériel  
pulmonaire protosystolique Arch mal coeur  
30 946 1937
- 940 LIAN C AND WELTI J J Le dédoublement du  
premier bruit Le premier bruit à préce ion  
auriculaire et le bruit de galop pré-systolique  
retardé Arch mal coeur 31 406 1938
- 941 LIAN C AND WELTI J J Flutter, ou mieux  
tremulation auriculaire et bradycardie par  
dilatation auriculo ventriculaire complète  
Auscultation et enregistrement graphique de bruits  
auriculaires Arch mal coeur 31 518 1938
- 942 LIAN C AND WELTI J J Les rythmes systoliques  
à trois temps l'étude étiologique suivie de  
remarques sur le sens que doit conserver l'ex-  
pression bruit de galop Acta Cardiol 5 103  
1950
- 943 LIAN C AND WELTI J J Les souffles pleuraux  
cardiopulmonaires J de méd et chir prat  
109 113 1935
- 943A LIAN C AND WELTI J J Le rythme sys-  
tolique à trois temps Étude étiologique suivie  
de remarques sur le sens que doit conserver  
l'expression bruit de galop Acta Cardiol 5  
109 1950
- 944 LIAN C WELTI J J DJORDJEVITCH AND STE-  
FANOVITCH Les souffles pleuraux cardiopul-  
monaires Arch mal coeur 30 412 1937
- 945 LIBMAN F AND SACKS B A hitherto unde-  
scribed form of valvular and mural endocarditis Arch  
Int Med 33 701 1934
- 945A LICHTENSTEIN L AND KAPLAN L Hereditary  
ochronosis Pathologic change observed in two  
necropsied cases Am J Path 30 99 1934
- 946 LICHTMAN S Diseases of the Liver Gall Bladder  
and Bile Ducts Ed 3 Lea & Febiger Philadel-  
phia 1933
- 947 LISKOFF W AND LEVINE S A Thyrotoxicosis as  
a cause of heart failure Am J Med Sc 206  
425 1943
- 948 LILJEBLAD C W BOBB J R R AND FISCHER  
M B Occurrence of endocarditis with valvular  
deformities in dogs with arteriovenous fistulae  
Proc Soc Exper Biol & Med 76 9 1950
- 949 LILJEBLAD C W CROCKETT J E AND DIMOND E C  
Ruptured congenital aneurysm of the sinus of  
Valvula Am Heart J 51 445 1956
- 950 LIND J AND WECHELUS C Human fetal circula-  
tion changes in the cardiovascular system at  
birth and disturbances in the postnatal closure  
of the foramen ovale and the ductus arteriosus  
Cold Spr Harb Symp Quant Biol 19 103  
1954
- 951 LINTON R R AND WHITE P D Arteriovenous  
fistula between the right common iliac artery  
and inferior vena cava report of a case and its  
occurrence following operation for ruptured  
intervertebral disk with cure by operation  
Arch Surg 50 6 1945
- 952 LINTZ R M Spontaneous mediastinal emphy-  
sema Arch Int Med 71 256 1933
- 953 LISSNER H H The whistled voice sound in

- and to the early diagnosis of pneumonic consolidation *M Rec* 96 412 1919
- 4 LEE W A Pericardial knock associated with spontaneous pneumothorax *Lancet* 1 1723 1928
- 5 LITTLE R C The cardiodynamic of tricuspid insufficiency *Proc Soc Exper Biol & Med* 68 607 1945
- 6 LITTLE R C Effect of atrial systole on ventricular pressure and closure of the A-V valves *Ann J Physiol* 166 209 1943
- 7 LITTLE R C Volume elastic properties of the right and left atrium *Ann J Physiol* 168 237 1945
- 8 LITTLE R C HILTON J C and SCHIFFERIN R D The first heart sound in normal and ectopic ventricular contractions *Circulation Res* 2 45 1954
- 9 LITVINSKY R Investigation of apparatus for registered phonocardiography according to the Mannheimer Standard system *Acta med Scandinav* 153 358 1949
- 10 LLOYD D SILVA J DILLON R F and CASSELL B M Syndrome of stenosis of the right pulmonary artery *Abstract Circulation* 16 911 1955
- 11 LONGE I B Emory University Co Personal communication
- 12 LUSCHKE W P and COLE O M Duration of the systole of the left ventricle of man *Ann J Physiol* 77 263 1926
- 13 LOWENSTAM L R Why are the segments of the semilunar valves three in number? *Clinic* 16 73 1915
- 14 LUTENBACH C and SCHLICKERFORD J Functional tricuspid incompetence in relation to the venous pressure *Brit Heart J* 19 303 1956
- 15 LOYCKE H QUICKER J J and ROSSMAN W L Sound produced at the pyloric sphincter bed *Proc* 12 91 1923
- 16 LOWENSTAM C A A valuable sign in the diagnosis of functional aortic insufficiency *Ann Int Med* 14 61 1940
- 17 LOWE H CANNON W F and LEVINE S A The syndrome of horizontal normal QRS complex and paroxysmal rapid heart action *Circulation* 11 697 1957
- 18 LUBAR J C and BIRN C C Spontaneous rupture of posterior papillary muscle of heart *Arch Pathol* 31 387 1931
- 19 LUDWIG C B Absorption and release of sound in solid tissue *J Acoust Soc Am* 22 847 1951
- 20 LUDWIG H Funktionellen Mitral stenosen durch Tumoren des linken Vorhofs *Zi chr klin Med* 123 587 1933
- 21 LUISADA A A Clinical applications of phonocardiography *Arch Pathol* 66 394 1937
- 22 LUISADA A A The diastolic sounds in normal and pathological conditions *Acta med Scandinav* 266 665 1957
- 23 LUISADA A A Discussion in Symposium on Cardiovascular Sound *Circulation* 16 270 and 411 1957
- 24 LUISADA A A The functional murmur the lasting to rest of a phant in *Chir* 21 570 1933
- 25 LUISADA A A The Heart Beat Graphic Methods in the Study of the Cardio Patient The Williams and Wilkins Co Baltimore 1953
- 26 LUISADA A A Decurrence of aortic aneurysm in horse *Vet Med* 38 245 1943
- 27 LUISADA A A On the apical systolic and diastolic murmurs in aortic regurgitation *Am Heart J* 28 157 1944
- 28 LUISADA A A On the pathogenesis of the sign of Traube and Duroziez in aortic insufficiency A graphic study *Am Heart J* 28 71 1947
- 29 LUISADA A A Symposium on Heart sound and murmurs held jointly with M.I. Buffalo Feb 1957
- 30 LUISADA A A Variable interval between electric and acoustic phenomena in aortic aortic filtration *Am Heart J* 23 715 1941
- 31 LUISADA A A and ALMURIC M M The systolic gallop rhythm *Acta cardiologica* 4 307 1941
- 32 LUISADA A A ALMURIC M M and LEVINE I Mechanism of production of first heart sound *Am J Physiol* 168 27 1947
- 33 LUISADA A A and ALMURIC C Phonocardiography as a clinical method of examination *Med Clin N America* 41 23 1957
- 34 LUISADA A A and FLETCHER F C Temporal relation between contraction of right and left side of the normal heart *Proc Soc Exper Biol & Med* 111 374 1941
- 35 LUISADA A A and CHASE C Clinical calibration in phonocardiography *Am Heart J* 28 407 1941
- 36 LUISADA A A HARRIS O M ALMURIC C CAMERON J and ZILL A B Murmurs in children a clinical and graphic study in 1000 children of school age *Ann Int Med* 48 307 1958
- 37 LUISADA A A HARRIS O M and ZILL A B Apical diastolic murmurs imitating mitral stenosis in graphic differentiation *Ann Int Med* 42 644 1955
- 38 LUISADA A A and LEE C H Simple method for recording intracardiac electrocardiogram and phonocardiogram during left or right heart catheterization *Am Heart J* 51 1957
- 39 LUISADA A A and ALMURIC C Early changes of mitral valve function in rheumatic heart disease *Am J Med* 15 23 1953
- 40 LUISADA A A and ALMURIC C The low frequency tracings of the precordium and epigastrum in normal and in leadcardiac patients *Am Heart J* 46 545 1957
- 41 LUISADA A A and MALTZER R Experimental

- studies on functional murmurs and extra sounds of the heart *Exper Med & Surg* 1 282 1943
- 901 LUISADA A A MENDOZA F AND ALMURUNG M M The duration of normal heart sound *Brit Heart J* 11 41 1949
- 902 LUISADA A A AND PEREZ MONTES L A phonocardiographic study of apical diastolic murmurs simulating those of mitral stenosis *Ann Int Med* 33 56 1950
- 903 LUISADA A A RICHMOND L AND ARVANIS C Selective phonocardiography *Am Heart J* 51 221 1956
- 904 LUISADA A A WEISS L AND HANTMAN H W A comparative study of electrocardiogram and heart sounds in common and domestic mammals *Cardiologia* 8 62 1944
- 905 LUISADA A A AND WOLFF L The significance of the pulmonary diastolic murmur in cases of mitral stenosis *Am J M Sc* 209 201 1945
- 906 LURIE P R Postural effects in tetralogy of Fallot *Am J Med* 16 297 1953
- 907 LUTFMACHER R De la sténose mitrale avec communication intraauriculaire *Arch mal coeur* 9 237 1916
- 908 LYNNWILFER C P AND DONAHOF J L Evaluation of innocent heart murmurs *South M J* 48 161 1955
- 909 LYON R A RAUB L W AND STIRLING J W Heart murmurs in newborn infants *J Pediatr* 16 310 1910
- 1000 LYONS R A AND KAPLAN S Patent ductus arteriosus in infancy *Pediatrics* 13 357 1954
- 1001 MAASS H AND WEBER A Herzchallregistrierung mittels differenzierender Filter Eine Studie zur Herzchallnormung *Cardiologia* 21 773 1952
- 1002 MACCALLUM J B On the muscular architecture and growth of the ventricles of the heart *Johns Hopkins Hosp Rep* 9 307 1904
- 1003 MACCALLUM W G Obliterative pulmonary arteriosclerosis *Bull Johns Hopkins Hosp* 49 37 1931
- 1004 MACCALLUM W G Rheumatic lesions of left auricle of heart *Bull Johns Hopkins Hosp* 35 329 1924
- 1005 MACCALLUM W C *Textbook of Pathology* Ed 17 p 702 W B Saunders Co Philadelphia 1912
- 1006 MACCURDY E ed *The Notebooks of Leonardo da Vinci* vol 1 p 277 Revival and Hitchcock New York 1938
- 1007 MACFARLAN D The acoustics of the stethoscope *J A M A* 110 2068 1939
- 1008 MACFARLANE M V SWAN W C A AND IRVINE R F (Newcastle upon Tyne) Personal communication
- 1009 MACKBY M J Cephalic bruit A review of literature and report of 6 cases *Am J Surg* 55 527 1942
- 1010 MACKENZIE J The intracranial bruit *Brain* 78 350 1955
- 1011 MACKENZIE J *Diseases of the Heart* Ed 2 p 231 Oxford Univ Press London 1910
- 1012 MACKENZIE J *Principles of Diagnosis and Treatment in Heart Affections* p 100 Oxford University Press London 1916
- 1013 MACKINNON J WADE L G AND VICKERS C F H Mitral stenosis with very high pulmonary vascular resistance and atypical features *Brit Heart J* 18 449 1956
- 1014 MACKLIN M T AND MACKLIN C C Malignant interstitial emphysema of the lungs and mediastinum is an important occult complication of many respiratory diseases and other conditions An interpretation of the clinical literature in the light of laboratory experiment *Medicine* 23 281 1944
- 1015 MACLEOD A G AND COHN A E A new piezo electric manometer to record intracardiac pressure *Am Heart J* 21 343 1941
- 1016 MACLEOD A C WILSON F N AND BARBER P S Observations on sound with particular reference to gallop rhythm and sounds of aortic origin *Proc Soc Exper Biol & Med* 29 1009 1931-1932
- 1017 MACMILLAN J I A Case in which both pulmonary veins emptied into persistent left superior vena cava *Canad M A J* 45 261 1941
- 1018 MACMILLAN J I S AND MORTON L A H Continuous venous hum in a case of portal cirrhosis *Brit Heart J* 17 105 1955
- 1019 MACWILLIAM J A AND MELVIN S The estimation of diastolic pressure in man and in vitro *Heart* 5 153 1913-1914
- 1020 MACRAE D J Spontaneous pneumomediastinum in pregnancy *Lancet* 1 907 1910
- 1021 MAGDELAINE I Contribution à l'étude des souffles cardio-pulmonaires (souffles diastoliques de la bête) *Paris thes* No 573 1897
- 1022 MAGNADIE F Mémoire sur l'origine des bruits normaux du cœur *Mém Acad roy des sciences de l'inst de France* 14 155 1835 (Communication read at the Académie de Sciences Feb 3 1834)
- 1022A MACHAFFEE D I SCHRAMM R AND CREECH O In Traumatic ventricular septal defect Report of a case treated successfully *J Louisiana M Soc* 109 321 1957
- 1023 MAHAIR I *Les Tumeurs et les Polypes du Cœur* Ma on & Cie Paris 1945
- 1024 MAHER J F MALLORY G H AND LAURENZ C A Rupture of the heart after myocardial infarction *New England J Med* 255 1 1956
- 1025 MAIER H C AND STOLT A P Congenital arteriovenous fistulas of thoracic wall *Circulation* 1 809 1950
- 1026 MALINER M M An unusual extracardiac murmur simulating organic heart disease Report of a case *Arch Pediatr* 53 496 1936
- 1027 MAJER M M AND OMNI I The significance of





- 1063 McILROY W D AND WHITELEY A H Relation of gas tension and hydrostatic pressure to intravascular bubble formation *Am J Physiol* 147 19 1946
- 1064 McILROY W D AND CRAY J Hepatic venous hum in cirrhosis of liver *J med* 2 1125 1953
- 1065 McINNIS S AND WHITE P D Acute cor pulmonale resulting from pulmonary embolism Its clinical recognition *J A M A* 104 1473 1935
- 1066 MCGREGOR M RAFAELORT M B SIRAGLE H B AND FRIEDMAN A J The calibration of heart sound intensity *Circulation* 13 252 1956
- 1067 MCGUIRE J AND McNAMARA R J Organic and relative insufficiency of the pulmonary valve *Am Heart J* 14 562 1937
- 1068 McKUSICK V A Carcinoid cardiovascular disease *Bull Johns Hopkins Hosp* 98 13 1956
- 1069 McKUSICK V A Chronic constrictive pericarditis I Some clinical and laboratory observations *Bull Johns Hopkins Hosp* 90 3 1952
- 1070 McKUSICK V A The diagnosis of organic mitral stenosis in the presence of sickle cell anemia *Am Heart J* 46 167 1953
- 1071 McKUSICK V A *Heritable Disorders of Connective Tissue* Mosby St Louis 1956
- 1072 McKUSICK V A Rheumatic stenosis of the mitral valve Report of a case with death over 4 years after valvulotomy *Arch Int Med* 95 557 1955
- 1073 McKUSICK V A Roussel of Paris and New Orleans I Experiments on the origin of the heart sound 125 years ago *Bull Hist Med* in press
- 1074 McKUSICK V A The study of mitral regurgitation by roentgen kymography with observations on the movement of cardiac excursions *Am J Roentgenol* 71 961 1953
- 1075 McKUSICK V A AND COCHRAN J H Constrictive endocarditis Report of a case *Bull Johns Hopkins Hosp* 90 90 1952
- 1076 McKUSICK V A AND COOLEY R N Drainage of right pulmonary vein into inferior vena cava Report of a case with radiologic analysis of the principal types of anomalous venous return from the lung *New England J Med* 252 291 1955
- 1077 McKUSICK V A HAHN D I BRAYSHAW J R AND HUMPHRIES J O Some hemodynamic effects of the Hufnagel operation for aortic regurgitation Studies in models and a patient *Bull Johns Hopkins Hosp* 95 332 1954
- 1078 McKUSICK V A AND HARVEY A M Disease of the pericardium *Advances Int Med* 7 157 1955
- 1079 McKUSICK V A JENKINS J T AND WEBB C N Iconic basis of the chest examination *Am Rev Tuberc* 72 12 1955
- 1080 McKUSICK V A KLINE F W AND WEBB C N Spectral phonocardiographic demonstrations of elected varieties of cardiovascular sound *Am Heart J* 49 911 1955
- 1081 McKUSICK V A LOCUS R B AND BAIRDSON H T Ascertaining of aortic valvular disease and aortic medial necrosis of the ascending aorta *Circulation* 46 158 1957
- 1082 McKUSICK V A, MASSENGALL O N JR WILSON M AND WEBB C N Spectral phonocardiographic studies in congenital heart disease *Brit Heart J* 18 403 1956
- 1083 McKUSICK V A MURRAY C I PETER R C AND WEBB C N Musical murmurs *Bull Johns Hopkins Hosp* 97 136 1955
- 1084 McKUSICK V A REAGAN W P SANTOS G W AND WEBB C N The splitting of heart sounds A spectral phonocardiographic evaluation of clinical significance *Am J Med* 18 519 1955
- 1085 McKUSICK V A TALBOT S A AND WEBB C N Spectral phonocardiography Problem and prospects in the application of the Bell sound spectrograph to phonocardiography *Bull Johns Hopkins Hosp* 94 187 1954
- 1086 McKUSICK V A WEBB C N BRAYSHAW J R AND TALBOT S A Spectral phonocardiography clinical studies *Bull Johns Hopkins Hosp* 95 90 1954
- 1087 McKUSICK V A WEBB C N HUMPHRIES J O AND REID J A On cardiovascular sound Further observation by means of spectral phonocardiography *Circulation* 11 819 1955
- 1088 McMITTAN I K R Aortic stenosis post mortem cinephotographic study of valve action *Brit Heart J* 17 56 1955
- 1089 McMITTAN I K R DALEY R AND MATTHEWS M B The movement of aortic and pulmonary valve studied post mortem by colour cinematography *Brit Heart J* 14 42 1957
- 1090 MEDA A AND SCARLETT C Phonocardiographic investigation of the variability of the first heart sound in auricular fibrillation *Cardiologia* 31 144 1957
- 1091 MEDD W I MATTHEWS M B AND THURSTFIELD W R R Clinician's disease *Thorax* 9 14 1954
- 1092 MILIK CERNASARIAN I A Zur Frage über die extra- und intraperikardialen Reibengeräusche *Ztschr Kreislaufforsch* 25 129 1953
- 1093 MIREN J F Courbe envelope des bruit du coeur *Arch mal coeur* 10 567 1956
- 1094 MIREN J F AND CARRARA J I Courbe envelope des bruit du coeur *Abstract Acta cardiologica* 181 1957
- 1095 MIZERO I A clinical study of three cases of primary tumor of the heart *Internat Clin* 4 331 1917
- 1096 MESSINGER C R Functional systolic murmurs in children *Am J Med* 217 71 1949
- 1097 MESSER A I COCHRAN T B RAFAELORT M B AND SIRAGLE H B The effect of cycle length on the time of occurrence of the first

- heart sound and the opening snap of mitral stenosis. *Circulation* 4 56 1951
- 1075 MEER A L MEER J W RASFAFORT M B AND SPRAGUE H H A study of the venous pulse in tricuspid valve disease. *Circulation* 1 34 1950
- 1076 MICHELON V Imitation of the tuning fork in the diagnosis of pulmonary disease. *Am J M* 72 13 1950
- 1077 MICHIGAN (U of) School of Public Health Theological Spectrum (Symposium) U of Michigan Press Ann Arbor 1950
- 1078 MIDDLETON F H CILBERT C B HEDGECOCK H H AND HESS C V Application of the phase principle of filtering to peccid phonocardiography. *IRE Transactions on Instrumentation* 10 1-15 June 1956
- 1079 MIZUKI A F AND HOLLOMAN K Beitrag zur Erklärung und diagnose von Bedeutung der Beckenstoma. *Archiv für die Naturgeschichte* 110 1951
- 1080 MILLER D C The Science of Mitral Stenosis The Macmillan Company New York 1957
- 1081 MILLER C AND POLLOCK B F Transcatheter pulmonary venous drainage. *Am Heart J* 49 17 1955
- 1082 MILLER M AND REEDER F M Fophagen phonocardiography. *Exper Med & Surg* 9 31 1950
- 1083 MILL J L Fugate and the Bell Van Los translation 1955 New York 1955
- 1084 MILLER W P AND BERTRAND C A The electrocardiogram in mitral stenosis. A study of 74 cases with observations on the R-R-V1 pattern. *Am J Med* 22 2-3 1957
- 1085 MINAKAWA T IMAI M AND YAMAGUCHI K Quantitative estimation of intensity of heart murmurs in children with valvular disease. *Tohoku J Exper Med* 38 25 1950
- 1086 MINNA K Chicago Peroral communication
- 1087 MILLER T A case of peripheral pulmonary stenosis. *Acta pathol* 42 300 1953
- 1088 MOLYNEUX L Recording low frequency phenomena on magnetic tape. *Electronics Engineering* 24 131 (March) 1952
- 1089 MONTGOMERY C M In Norm and Pathology Diseases of the Heart and the Circulation of Physiology and Pathology W C Saunders Company Philadelphia 1958
- 1090 MOORE J F The Mode and Treatment of Syphilis p 283 Charles C Thomas Springfield 1947
- 1091 MORTON P R SCHWARTZ M L ARNETT S R AND SMITH H W AND REID M V A phonocardiographic study of mitral valvular disease complicated by auricular fibrillation. An analysis of the factors responsible for variations in the time of occurrence of the first heart sound and the opening snap. *Cardiology* 29 150 1956
- 1092 MURPHY J W The Stenosis of the Aorta. Translated from the Latin by Benj Alexander M D London 1761 Book II On Diseases of the Thorax Letter 16 article 21 p 391
- 1093 MURPHY J H AND BRUNNELL H B Ventricular septal defect simulating patent ductus arteriosus. *Proc Staff Meet Mayo Clin* 25 69 1950
- 1094 MURPHY J H Peroral communication
- 1095 MURPHY J H AND STARK T V Isolated pulmonary valvular regurgitation. *Circulation* 14 3069 1956
- 1096 MOSCOWITZ H I DODD J F CRIP J J AND WELLSCHWITZ W Intracardiac phonocardiography: correlative study of mechanical, acoustic and electroacoustic in experimental valvular stenosis. *Circulation* 16 918 1957
- 1097 MOSCOWITZ H I CORCORAN J J BRUNSWOLD F ANDERSON S S SERRA S O FARRAR H I HUMPHREYS A AND RAVICH M M The use of simultaneous left heart pressure and measurement in evaluating the effects of mitral valve surgery. *Am J Med* 18 111 1955
- 1098 MORTON W C AND JOHN A V Differential effects of stretch upon the stroke volumes of the right and left ventricle. *Am J Physiology* 139 57 1951
- 1099 MORTON H I COURVANT A WERRO I HUMPHREYS A AND RAVICH M M The influence of the period of induced acute anoxia upon pulmonary artery pressure in man. *Am J Physiology* 150 715 1957
- 1100 MORTON H I The early diastolic rumble of mitral stenosis. *Brit Heart J* 17 147 1957
- 1101 MORTON H I Local precordial bulbar murmurs. *Am J Med* 12 140 1952
- 1102 MORTON H I The opening snap of mitral stenosis. *Brit Heart J* 15 113 1955
- 1103 MORTON H I Precordial bulbar murmurs. *Brit Heart J* 19 31 1957
- 1104 MORTON H I AND BRUNNELL H B The typical mitral murmur in mitral stenosis. *Brit Heart J* 16 255 1954
- 1105 MOVITT I H AND CERNY H Pure mitral insufficiency of rheumatic origin in adults. *Ann Int Med* 38 951 1953
- 1106 MOVITT J H AND ACKERMAN A J Hereditary hemorrhagic telangiectasia associated with pulmonary arteriovenous fistula in two members of a family. *Ann Int Med* 23 75 1951
- 1107 MOYER J J AND DICHOAL I Pfuhe electrophonocardiographic du rythme de rétrogradation mitral. *Arch mal coeur* 74 1950
- 1108 MOYER J J AND DICHOAL I Le rythme de rétrogradation dans la maladie de Stokes-Adams. *Enregistrements électrophonocardiographiques*. *Arch mal coeur* 23 24 1950
- 1109 MURPHY J C AND WATTS J D The auscultatory gap in sphygmomanometry. *Arch Int Med* 41 243 1952

- 1063 McElroy, W. D. and Whiteley, A. H. Relation of gas tension and hydrostatic pressure to intravascular bubble formation. *Am J Physiol* 147: 19, 1946.
- 1064 McFadden, A. J. S. and Cray, J. Hepatic venous hum in cirrhosis of liver. *Lancet* II: 1128, 1953.
- 1065 McGinn, S. and White, P. D. Acute cor pulmonale resulting from pulmonary emboli in its clinical recognition. *J A M A* 104: 1473, 1935.
- 1066 McGreer, M. Rappaport, M. B. Siragusa, H. H. and Friedlich, A. I. The calibration of heart sound intensity. *Circulation* 13: 252, 1956.
- 1067 McGuire, J. and McNamara, R. J. Organic and relative inefficiency of the pulmonary valve. *Am Heart J* 14: 502, 1937.
- 1068 McKusick, V. A. Carcinoid cardiovascular disease. *Bull Johns Hopkins Hosp* 98: 13, 1956.
- 1069 McKusick, V. A. Chronic constrictive pericarditis. I. Some clinical and laboratory observations. *Bull Johns Hopkins Hosp* 90: 3, 1952.
- 1070 McKusick, V. A. The diagnosis of organic mitral stenosis in the presence of sickle cell anemia. *Am Heart J* 45: 467, 1953.
- 1071 McKusick, V. A. *Heritable Disorders of Connective Tissue*. Mo. by St. Louis, 1956.
- 1072 McKusick, V. A. Rheumatic stenosis of the mitral valve. Report of a case with death over 4 years after valvulotomy. *Arch Int Med* 95: 557, 1955.
- 1073 McKusick, V. A. Rousselot of Paris and New Orleans. Experiments on the origin of the heart sound 125 years ago. *Bull Hist Med* in press.
- 1074 McKusick, V. A. The study of mitral regurgitation by roentgen kymography with observations on the movement of cardiac calcifications. *Am J Roentgenol* 71: 961, 1954.
- 1075 McKusick, V. A. and Cochran, T. H. Constrictive endocarditis. Report of a case. *Bull Johns Hopkins Hosp* 99: 90, 1952.
- 1076 McKusick, V. A. and Coffey, R. N. Drainage of right pulmonary vein into inferior vena cava. Report of a case with radiologic analysis of the principal types of anomalous venous return from the lung. *New England J Med* 252: 291, 1955.
- 1077 McKusick, V. A., Hahn, D. I., Brayshaw, J. R. and Humphries, J. O. Some hemodynamic effects of the Hufnagel operation for aortic regurgitation. Studies in model and a patient. *Bull Johns Hopkins Hosp* 95: 332, 1954.
- 1078 McKusick, V. A. and Harvey, A. M. Disease of the pericardium. *Advances Int Med* 7: 157, 1955.
- 1079 McKusick, V. A., Jenkins, J. T. and Webb, C. N. Acoustic basis of the chest examination. *Am Rev Tuberc* 72: 12, 1955.
- 1080 McKusick, V. A., Kline, F. W. and Webb, C. N. Spectral phonocardiographic demonstrations of selected varieties of cardiovascular sound. *Am Heart J* 49: 911, 1955.
- 1081 McKusick, V. A., Loefer, R. B. and Bannock, H. T. Association of aortic valvular disease and cystic medial necrosis of the ascending aorta. *Circulation* 46: 188, 1957.
- 1082 McKusick, V. A., Massengale, O. N., Jr., Wilson, M. and Webb, C. N. Spectral phonocardiographic studies in congenital heart disease. *Brit Heart J* III: 403, 1956.
- 1083 McKusick, V. A., Murray, G. F., Pfeiffer, P. C. and Webb, C. N. Musical murmur. *Bull Johns Hopkins Hosp* 97: 136, 1955.
- 1084 McKusick, V. A., Reacan, W. P., Santos, C. W. and Webb, C. N. The plotting of heart sounds. A spectral phonocardiographic evaluation of clinical significance. *Am J Med* 29: 819, 1955.
- 1085 McKusick, V. A., Talbot, S. A. and Webb, C. N. Spectral phonocardiography. Problem and prospects in the application of the Bell sound spectrograph to phonocardiography. *Bull Johns Hopkins Hosp* 94: 187, 1954.
- 1086 McKusick, V. A., Webb, C. N., Brayshaw, J. R. and Talbot, S. A. Spectral phonocardiographic clinical studies. *Bull Johns Hopkins Hosp* 95: 90, 1954.
- 1087 McKusick, V. A., Webb, C. N., Humphries, J. O. and Reid, J. A. On cardiovascular sound. Further observation by means of spectral phonocardiography. *Circulation* 11: 819, 1955.
- 1088 McMullan, I. K. R. Aortic stenosis: a post mortem cinephotographic study of valve action. *Brit Heart J* 17: 56, 1955.
- 1089 McMullan, I. K. R., Daley, R. and Matthews, M. B. The movement of aortic and pulmonary valve studied post mortem by colour cinematography. *Brit Heart J* 14: 42, 1952.
- 1090 Mfeda, A. and Scavetti, C. Phonocardiographic investigation of the variability of the first heart sound in atricular fibrillation. *Cardiologia* III: 144, 1957.
- 1091 Mfeda, W. L., Matthews, M. B. and Thursfield, W. R. R. Fibrous disease. *Thorax* 9: 14, 1954.
- 1092 Melik, C. A. N. I. A. Zur Frage über die extra- und intrapericardialen Reibegeräusche. *Ztchr Aerzheilkunde* 25: 129, 1933.
- 1093 Merle, J. F. Courbe enveloppe des bruits du coeur. *Arch mal coeur* 49: 567, 1956.
- 1094 Mfelen, J. T. and Cahner, J. F. Courbe enveloppe de bruit du coeur. Abstract. *Acta cardiologica* 14: 157, 1957.
- 1095 Méroz, I. A clinical study of three cases of primary tumor of the heart. *Internat Clin* 4: 331, 1917.
- 1096 Messinger, C. R. Functional systolic murmurs in children. *Am J Sc* 217: 71, 1919.
- 1097 Messer, A. I., Coleman, T. B., Rappaport, M. H. and Sprafkin, H. B. The effect of cycle length on the time of occurrence of the first

- 1164 OWEN S AND EAST T Rupture of an aortic mitral valve into right side of the heart *Brit Heart J* 17 541 1955
- 1165 OWEN S (1) The genesis of heart murmur *New England J Med* 241 133 1949
- 1166 OWEN S AND BRAIN MEYER F *The Heart & its Normal and Pathological Conditions* Oxford University Press London 1953
- 1167 OWEN T The sound produced by the friction of normal aortic valve *Am Heart J* 17 643 1939
- 1168 OWEN W On a remarkable heart murmur heard at a distance from the chest wall *Med Times & Gaz* 2 477 1880
- 1169 OWEN W On the aortic diastolic murmur of children *Boston Med & Surg J* 103 29 1880
- 1170 OWEN W Sir Thomas Browne in his *Medicine & its Art* *Biographical Memoirs* 11 1 p 256 Oxford University Press 1905
- 1171 OWEN ON P J CALLAHAN J S AND HERNARD J F Mitral insufficiency from ruptured chordae tendineae simulating aortic stenosis *Proc Staff Meet Mayo Clin* 33 235 1948
- 1172 OXFORD B Familial amyloidosis of the meninges *Verh u Koninklijke Akad 30* 105 1940
- 1173 OWEN S C AND WOOD P A new method of determining the degree or absence of mitral obstruction *Brit Heart J* 17 41 1955
- 1174 OWEN W R THOMAS W A CLEMENS B AND BRADY F (Recognize embolism in pulmonary embolism) *New England J Med* 240 119 1949
- 1175 PADGETT H Origins heart lesion of foetus in utero *South Afric J* 18 315 1951
- 1176 PALFREY F W In cultivation of Corynebacterium *New England J Med* 247 1 1952
- 1177 PALFREY F W The aortic of the first heart sound *New England J Med* 200 117 1929
- 1178 PALFREY F W AND AYER J B A case of chronic infective endocarditis with ulcerated aortic chordae tendineae *Boston M & S J* 187 55 1912
- 1179 PALFREY R S AND WHITE P D A note on the continuous humming murmur heard in the supra and infraorbital foramen over the maxillary foramen in children *New England J Med* 199 129 1928
- 1180 PARDEE H E B Chairman Criteria Committee on aortic stenosis *Diagnosis of Diseases of Heart and Blood Vessels* New York Heart & Vessels
- 1181 PARDEE H E *De cordis et sanguinis motione ad interiora et contra eum* 1633 (Often reprinted; Quoted by Darcourt)
- 1182 PARDEE F D New thoracic murmur with two new instruments *The refractoscope and the partial stethoscope* *J Exper Med* 100 607 1918
- 1183 PARKER F Wound of the oesophagus with perforation of the pericardium *Tr Bath Soc Lond* 2 40 1849
- 1184 PARKER A J AND HERNIMAN S Heart sound failure & phonocardiographic study of this phenomenon in acute coronary occlusion *J Am M A* 96 111 1931
- 1185 PARKER C Ventricular extension into the abdominal wall *Brit Heart J* 11 31 1951
- 1186 PARKER J S AND HERNIMAN B W The aortic and pulmonary arteries in Fallot's tetralogy *Brit Heart J* 11 31 1951
- 1187 PARKER C *Anders medicorum full et mem* *Med Hist J* 2 (1919) 1931
- 1188 PARKER H R HERNIMAN M OXFORD J S AND NAYAK S S Congenital complete atrioventricular block a hemodynamic and clinical study *Circulation* 18 981 1958
- 1189 PARKER C KERNAN J S AND BROWN S One main type of cardiovascular disease associated with free aortic regurgitation into the heart *Am J Circulation* 18 914 1958
- 1190 PARKER S AND WHITE P H AND WHITE P D Coronary arteriovenous fistula *Am Heart J* 37 411 1949
- 1191 PARKER S AND MANNHEIMER L The physiological heart murmur in children *Acta Paediatr* 46 45 1957
- 1192 PARKER J Die Entstehung und klinische Bedeutung des Calcipylasmus des Herzes *Arch Klin Med* 64 10 1908
- 1193 PARKER J Leber relative Inuffizienz der funktionell vergrößerten Mitralventil *Arch Klin Med* 100 19 1915
- 1194 PARKER P *Über die Entstehung gestaute Thrombus oder wasserigen Herzes* *Ztschr Klin Med* 28 452 1843
- 1195 PARKER J H Case of diastolic heart murmur heard during life *Tr Bath Soc Lond* 10 335 1850
- 1196 PARKER T B *Half century of the Heart* *Heart J Churchill* London 1954 (1 2 1956)
- 1197 PARKER T B Report on case of dilating aneurysm *Tr Bath Soc Lond* 14 5 1843
- 1198 PARKER T B Late stage Disease of the Heart *J C & Churchill* 111 London 1963
- 1199 PARKER H S Aypical diastolic murmurs in iron anemia *Brit Heart J* 11 95 1951
- 1200 PARKER J D OXFORD J S AND THOMAS K B Occluding thrombus of the right atrium Intermittent tricuspid occlusion in a case of atrial infarction with mural thrombus *Am J Med* 22 131 1957
- 1201 PARKER F The translocation of sound by the femur *Clin J* 17 109 1915
- 1202 PARKER J H KERNAN J W AND WOOD F H Interatrial pressure relation hips after closure of atrial septal defects in man *Circulation* 18 508 1958

- 1131 Muir D C and Brown J W Congenital heart disease Brit M J 1 966 1935
- 1132 Müller O and Shillingford J The blood flow in the right atrium and superior vena cava in tricuspid incompetence Brit Heart J 17 163 1955
- 1133 Müller O and Shillingford J Tricuspid incompetence Brit Heart J 16 196 1954
- 1134 Muscrove J I and MacQuinn R I Successful treatment of air embolism J A M A 150 25 1952
- 1135 Myers G S Scannell J C Waman S M Demond E G and Hurst J W Atypical patent ductus arteriosus with absence of the usual aortic pulmonary pressure gradient and of the characteristic murmur Am Heart J 41 919 1951
- 1136 Myers J D Murdoch H V McIntosh H D and Blaisdell R K Observations on continuous murmurs over partially obstructed arteries: An explanation for the continuous murmur found in the aortic arch syndrome Arch Int Med 97 726 1956
- 1137 Nadas A S Pediatric Cardiology W B Saunders Co Philadelphia 1957
- 1138 Nadas A S and Armstrong M M Atrial diastolic murmurs in congenital heart disease of Lutembacher's syndrome Am Heart J 43 601 1952
- 1139 Nazzi V Riccio C and Mada A Considerations sur la dynamique du coeur à systole ventriculaire (étude au moyen de la méthode polygraphique Cardiologia 11 319 1961
- 1140 Neill C Baltimore Personal communication
- 1141 Neill C and Mounsey P Auscultation in patent ductus arteriosus with a description of two fistulae simulating patent ductus Brit Heart J 20 61 1958
- 1142 Newman Kleinschal K and Steffen H Arch Tierheill 111 401 1935
- 1143 Nichols J W McK Subacute bacterial endocarditis Practitioner 107 421 1921
- 1144 Nichols H T Likoff W Goldberg H and Fitch M The genesis of the presystolic murmur in mitral stenosis Am Heart J 52 379 1956
- 1145 Niemeyer F von A Textbook of Practical Medicine Vol 1 p 393 Translated by C H Humphreys and C L Hackley D Appleton and Co New York 1862
- 1146 Nielsen V N J and Kramer K Stromvolum pulc der herznähen Venen bei verschiedenen Kreislaufzuständen Ztchr Biol 106 346 1964
- 1147 Nörk J Das Elektrokardiogramm der Pferde Seine Aufnahme und Form Ztchr Biol 61 197 1913
- 1148 Norris C W and Iandis H R M Diseases of the Chest and the Principles of Physical Diagnosis Ld 6 p 147 W B Saunders Company Philadelphia 1935
- 1149 Nuzzum F R Auscultatory evidence of cardiac rupture Am Heart J 39 909 1950
- 1150 Nygaard K K Wilder M and Berkson J Relation between velocity of blood and relative volume of erythrocyte Am J Physiol 114 175 1935
- 1151 Nye C and Björck C Phonocardiograms of aortic murmur from a case of mitral stenosis and heart block Brit Heart J 10 16 1948
- 1152 Obrastrow W I Ueber die verdoppelten und akzentuierten Herztonen bei unmittelbarer Kommunikation des Herzens Ztchr klin Med 87 70 1900
- 1153 O'Brien K H and Fabricius J Pulmonary valvular regurgitation during twenty seven years after gonorrheal endocarditis Am Heart J 52 791 1956
- 1154 O'Brien K H and Warren I Fourteen cases of loud apical aortic murmur in patient under 30 years A follow up study Acta Cardiol 11 164 1956
- 1155 Oliver B M A rooster for videography Proc I R F 38 1301 1950
- 1156 Oliver F J Murmurs from the record of the Boston Society for Medical Improvement Investigation of Mitral Leaflet motion appearance (Findings of H C Clark Ill and Shaw) Boston Med Surg J 111 480 1905
- 1157 Olivier J Le Dr Frangco Mior de Cencire (1779-1843) et la découverte de bruit du coeur foetal Rev med Suisse Rom 65 481 1945
- 1158 Olney M D and Stephens H B Correlation of aortic in children J Pediatr 37 639 1950
- 1159 Olson H F Musical Engineering An Engineering Treatment of the Interrelated Subjects of Speech Music Musical Instruments Acoustics Sound Reproduction and Hearing McGraw Hill New York 1952
- 1160 O'Malley C D and Sander C M Leonardo da Vinci on the Human Body Henry Schuman New York 1952
- 1161 Ongley I A Symposium on cardiovascular sound Circulation 16 431 1957
- 1162 Ongley I A Siraque H B and Rappaport M H The diastolic murmur of mitral stenosis New England J Med 253 1049 1955
- 1163 Osheshtin A K On intracardiac pressure measurement J Acous Soc Amer 29 1144 1957
- 1164 Osheshtin L H A section of adult type contraction of the aorta with endocardial fibroelastosis in infancy Bull John Hopkins Hosp 93 309 1953
- 1165 Osheshtin M J Durant T M and Lynch P Body position in relation to venous air embolism and aortic endocardial reperforatory changes Am J M Sc 225 362 1955



- 1131 Muir D C and Brown J W Congenital heart disease Brit M J 1 966 1935
- 1132 Muller O and Shillingford J The blood flow in the right atrium and superior vena cava in tricuspid incompetence Brit Heart J 17 163 1955
- 1133 Muller O and Shillingford J Tricuspid incompetence Brit Heart J 16 195 1954
- 1134 Muscrove J E and MacQuinn R I Successful treatment of air embolism J A M A 150 28 1952
- 1135 Myers G S Scannell J G Wyman S M Diamond E G and Hurst J W Atypical patent ductus arteriosus with absence of the usual aortic pulmonary pressure gradient and of the characteristic murmur Am Heart J 41 619 1951
- 1136 Myers J D Mordalich H V McIntosh H D and Blaisdell R K Observations on continuous murmurs over partially obstructed arteries An explanation for the continuous murmur found in the aortic arch syndrome Arch Int Med 97 726 1956
- 1137 Nadas A S Pediatric Cardiology W B Saunders Co Philadelphia 1957
- 1138 Nadas A S and Atimurinc M M Atypical diastolic murmurs in congenital heart disease rarity of Lutembacher's syndrome Am Heart J 41 691 1952
- 1139 Nazzi V Riccio C and Meda A Contractions sur la dynamique du coeur La systole ventriculaire (étude au moyen de la méthode polygraphique Cardiologia 24 319 1954)
- 1140 Nebel C Baltimore 1st oral communication
- 1140A Nebel C and Mounsey P An cultivation in patent ductus arteriosus with a description of two fistulae simulating patent ductus Brit Heart J 20 61 1958
- 1141 Neumann Kleinspali K and Steffan H Arch Tierheilk 69 401 1935
- 1142 Nichols J W McK Subacute bacterial endocarditis Practitioner 107 424 1921
- 1143 Nichols H T Ikkoff W Goldberg H and Fuchs M The genesis of the presystolic murmur in mitral stenosis Am Heart J 52 379 1956
- 1144 Niemeyer F von 1 Textbook of Practical Medicine v 1 p 393 Translated by C H Humphreys and C E Hackles D Appleton and Co New York 1862
- 1145 Nilsson V N J and Kramer K Stromvolumpulser der herznahen Venen bei verschiedenen Kreislaufzuständen Ztschr Biol 106 386 1954
- 1146 Norris J Das Elektrokardiogramm des Herdes Seine Aufnahme und Form Ztschr Biol 61 197 1913
- 1147 Norris G W and Landis H R M Diseases of the Chest and the Principles of Physical Diagnosis 1d 6 p 147 W B Saunders Company Philadelphia 1938
- 1148 Nyman I R Auscultatory evidence of cardiac rupture Am Heart J 40 903 1950
- 1149 Nygaard K K Widler M and Branson J Relation between viscosity of blood and relative volume of erythrocytes Am J Physiol 114 18 1935
- 1150 Nyman C and Björck C Phonocardiograms of aortic murmurs from a case of mitral stenosis and heart block Brit Heart J 9 16 1947
- 1151 Obrastzow W P Ueber die verdoppelten und doppelten Herztöne bei unmittelbarer Auskultation des Herzen Ztschr klin Med 87 70 1905
- 1152 Olsen K H and Farnick J Pulmonic valvular regurgitation during twenty seven years after gonorrheal endocarditis Am Heart J 60 791 1956
- 1153 Olsen K H and Wabers E Fourteen cases of loud apical systolic murmurs in patients under 30 years a follow up study Acta cardiologica 11 164 1956
- 1153A Oliver B M A router for video signals Proc IRE 38 1301 1950
- 1154 Oliver F I Extract from the record of the Boston Society for Medical Improvement Examination of Mages to mortem appearance (Findings of H C Clark Mlt and Shaw) Boston Med & Surg J 58 480 1858
- 1155 Olivier J Le Dr Fringot Mayor de Genève (1779-1854) et la découverte des bruits du coeur foetal Rev med Suisse Rom 481 1945
- 1156 Olney M D and Stephens H M Correlation of systolic children J Pediatr 37 639 1950
- 1157 Olson H I Musical Engineering An Engineering Treatment of the Interrelated Subjects of Speech Music Musical Instruments Acoustics Sound Reproduction and Hearing McGraw Hill New York 1952
- 1158 O'Malley C D and Saunders G M Leonardo da Vinci on the Human Body Henry Schuman New York 1952
- 1159 Ongley E A Symposium on cardiovascular sound Circulation 16 431 1957
- 1160 Ongley E A Sprague H B and Raffaport M B The diastolic murmur of mitral stenosis New England J Med 253 1049 1955
- 1161 Oppenheim A K On intrinsic pressure measurement J Acoust Soc of Amer 23 1144 1957
- 1162 Oppenheimer L H A question of adult type coarctation of the aorta with endocardial fibroelastosis in infancy Bull Johns Hopkins Hosp 93 309 1953
- 1163 Oppenheimer M J Durant T M and Lynch P Body position in relation to venous pressure and aortic endocardial culture peripartory change Am J M Sc 225 567 1957

- clinical measurements of arterial blood pressure with a note on an cuffatory grip Bull Johns Hopkins Hosp 69 501 1941
- 1710 RAY W F JR BARBARIE J I ELLERED I M AND HUNN F L Local and long distance transmission and storage of electrocardiograms and other low frequency signals Circulation Re 1 218 1953
- 1711 RAPPAPORT M B AND SPRAGUE H B Indirect pharyngomanometry J Lab & Clin Med 23 635 1943
- 1712 RAPPAPORT M B AND SPRAGUE H B The effect of improper fitting of tethoscope to ears on auscultatory efficiency Am Heart J 43 713 1952
- 1713 RAPPAPORT M B AND SPRAGUE H B The effects of tugging force on tethoscope efficiency Am Heart J 42 605 1951
- 1714 RAPPAPORT M B AND SPRAGUE H B The graphic registration of the normal heart sound I A graphic analysis of the normal heart sound Am Heart J 23 501 1947
- 1715 RAPPAPORT M B AND SPRAGUE H B The isoslope and physical law which govern auscultation and their clinical application The auscultoscope and the electrical amplifying tethoscope and tethograph Am Heart J 21 220 1941
- 1716 RAYNE W H Spontaneous rupture of the aorta into the superior vena cava: a case report with some matters for establishing the diagnosis Acta Cardiol 4 260 1949
- 1717 RAYN A Auscultation of the Heart (Chicago) Year Book Publishers 1958
- 1718 RAYN A AND BRIDGER F The significance of the first heart sound in auricular fibrillation with mitral stenosis Am Heart J 41 339 1951
- 1719 RAYN A AND DARTER W Atrial diastolic murmurs in patent ductus arteriosus Ann Int Med 30 303 1948
- 1720 RAY H C On the external system of the common Indian rat snake (*Naja mucosa* (Linn)) J Morph 66 331 1934
- 1721 RAYNALD R AND MARSHALL F C Thromboses de l'oreillette droite déterminants une infarctus embolique en infarctus pulmonaire et une infarctus rétro-aortique (epithélio) Ann Med Chir 4 34-41 Feb 1933
- 1722 REAF J S AND FLETCHER W B The effects of artificial pressure in the chest on the electrical rhythm Am J Med 19 177 1955
- 1723 REAGAN J A AND HENNINGER J O Sympathetic stimulation of the vagus nerve in the treatment of chronic angina pectoris Bull Johns Hopkins Hosp 97 1 1952
- 1724 REAGAN J A O Dilatation of the aorta due to granulomatous (great cell) aortitis Brit Heart J 19 706 1957
- 1725 REIFENSTEIN E H HOGG R M AND WALTER H H Clinical and physiologic studies in Fabry's malformation Clin Res 4 91 1956
- 1726 REINARTS C H JENSEN S A AND COHEN H E Coarctation of the aorta: A review of 101 autopsied cases of the adult type - a study of age and older Am Heart J 23 116 1947
- 1727 REINHOLD J D AND VALLAN A N The role of an aortic catheter in the diagnosis of congenital heart disease a phonocardiographic study of children Am Heart J 47 405 1954
- 1728 REINHOLD J D AND VALLAN A N Familial hypoplastic anemia with congenital dilatation of the aorta (Pancost's anemia) Blood 7 915 1952
- 1729 REINHOLD J AND REINECKE A Relation of the first and second heart sound to the cardiac cycle Brit Heart J 19 173 1957
- 1730 REINHOLD J REINECKE A AND RICHARDSON R L The heart sound and the arterial pulse in congenital aortic stenosis Brit Heart J 17 377 1955
- 1731 REINHOLD J W HAMILTON W F AND ALQUIST R L Interrelation between the length of a stroke volume and left ventricular work in the dog Am J Phys 154 6 1948
- 1732 REINIERE J Sécheresse de l'aorte et de la valve mitrale gauche. Anémie du cœur diagnostiquée par la valve Bull Med 10 483 1948
- 1733 REINIERE J AND STANLEY J Acquired diverticulum of mitral valve Brit M J 3 912 1953
- 1734 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1735 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1736 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1737 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1738 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1739 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1740 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1741 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1742 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1743 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1744 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1745 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1746 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1747 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1748 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1749 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947
- 1750 REISS W H AND KRIEGER C S Dissecting aneurysm with signs of aortic insufficiency J A M A 120 1297 1947



- 1200 PERLOFF, J. Second symposium on cardiovascular sound. Circulation in press.
- 1201 PERLOFF, J. K. AND HARVEY, W. P. Mechanism of murmur of tricuspid stenosis. Abstract Circulation 111 925 1957
- 1202 PERLOFF, J. K. AND HARVEY, W. P. Mechanisms of fixed splitting of the second heart sound. Abstract Circulation 111 924 1957
- 1203 PETERSON, A. I. G. AND BERANKE, L. L. *Handbook of Noise Measurement*. General Radio Co. Cambridge Mass. 1954
- 1204 PETIT, A. *Traité de Médecine de Charcot*. Bouche and Briand Paris 1902
- 1205 PHEAR, A. C. On the presystolic apex murmur without mitral stenosis. *Lancet* 2 716 1895
- 1206 PHELIPPEAU, Archives de Tocologie des Maladies des Femmes et des Enfants Nouveau Né 304 1879
- 1207 PHILIPSON, J. AND SALTZMAN, C. F. Combined ventricular septal defect and aortic insufficiency. *Acta radiol* 44 269 1955
- 1208 PICKERING, C. W. *High Blood Pressure*. Grune & Stratton New York 1954
- 1209 PICKENY, M. M. Mediastinal emphysema and idiopathic spontaneous pneumothorax. *Virginia M. Month* 111 315 1941
- 1210 PINTO, I. AND RODRIGUEZ, S. A study of the acoustic findings in patent ductus arteriosus. *Cardiologia* 28 1 1956
- 1211 PIORRY, P. A. *De la percussion médiate et des signes obtenus à l'aide de ce nouveau moyen d'exploration dans les maladies des organes thoraciques et abdominaux*. Chaudé et Baillière Paris 1828
- 1212 PITTS, C. N. On the case of Friedrich Schlegel. Its clinical history and postmortem appearance. *Cum. Hosp. Rep.* 11 369 1847
- 1213 PLACHTA, A. AND SPFFER, F. D. The coexistence of rheumatic heart disease and sickle cell anemia. *Am J Clin Path* 22 970 1952
- 1214 PLESCH, J. *Die Herklappenfehler. Spezielle Pathologie und Therapie innerer Krankheiten (Kraus & Brugsch)*. Urban & Schwarzenberg Berlin 4 II 1001 1955
- 1215 PRESSER, M. S. The dynamics of cavitation bubble. *J Appl Mech* 16 277 1949
- 1216 POMERENKE, W. T. AND BISHOP, F. W. Amplification of fetal heart sound. *Am J Obst & Gynec* 55 851 1938
- 1217 POPPER, J. L. Crural bruit—its significance. *S. Clin North America* 36 881 1955
- 1218 LOFFER, H. KUSHNER, D. S. AND CASLER, B. Adult fibroelastosis with congenital tricuspid stenosis. *Circulation* 14 412 1956
- 1219 PORTER, W. B. Case of gonococcal endocarditis with rupture of the aortic valve and death from acute pulmonary edema. *Heart* 16 201 1933
- 1220 PORTER, W. B. Diaphragmatic flutter with symptoms of angina pectoris. *J. A. M. A.* 106 992 1936
- 1221 PORTER, W. B. The syndrome of rupture of an aortic aneurysm into the pulmonary artery. *Am Heart J* 23 468 1942
- 1222 PORTER, W. B. AND JAMES, G. W. III. The heart in anemia. *Circulation* 8 111 1953
- 1223 POTAIN, P. C. *Bull. Soc. Anat. de Paris* Aug 25 1886. Quoted by P. Mounier. *Brit Heart J* 17 143 1955 (ref 1122)
- 1224 POTAIN, I. C. *Clinique médicale de la Charité*. Masson Paris 1894
- 1225 POTAIN, P. C. Du rythme cardiaque appelé bruit de galop de son mécanisme et de sa valeur étiologique. *Bull. et mém. Soc. méd. hôp. Paris* 12 137 1875
- 1226 POTAIN, I. C. Note sur le doublement normal du bruit du cœur. *Bull. et mém. Soc. méd. hôp. Paris* p. 135 1866
- 1227 POTAIN, P. C. Quelques recherches sur le bruit ventriculaire anormal qui survient dans les hémorrhagies. *Paris thet* 1853
- 1228 JOTT, *Biography of Theodor Weber*. *München med. Wchnschr* 45 1089 1899
- 1229 POTTER, R. K. Introduction to technical discussion of sound portrayal. *J. Acoust. Soc. Am* 18 1 1946
- 1230 POTTER, R. K., KOPF, C. A. AND CRISP, H. C. *Visible Speech*. D. Van Nostrand Co. Inc. New York 1947
- 1231 POWELL, M. I. AND HILLER, H. C. Pulmonary correction. *Med J Australia* 1 272 1955
- 1232 POWELL, S. J. Dissection of the left ventricle. Case report with special reference to electrocardiographic findings. *Am Heart J* 55 515 1958
- 1233 PRECK, J. AND CASSIDY, D. I. Dye dilution curve and cardiac output in newborn infants. *Circulation* 11 799 1955
- 1234 PRECK, O., KATZ, L. N., BENNETT, I., ROSENMAN, R. H., FISHMAN, A. P. AND HWANG, W. Determination of kinetic energy of the heart in man. *Am J Physiol* 159 481 1949
- 1235 PRECK, O., WOOD, F. JR. AND JULIAN, O. C. Diastolic murmur of the tricuspid valve hyperdynamic murmurs in patient with atrial septal defect and murmur of organic tricuspid stenosis. *Circulation* 12 761 1955
- 1236 PROLDITZ, W. I. AND MCCORMACK, I. J. Rupture of the aortic valve. *Circulation* 11 750 1955
- 1237 QUAIN, *Anatomy*. Ed. H. Vol. IV. The Heart. London 1929 (Quoted by McMillan (1973))
- 1238 QUINTAD, C. AND STEINERT, R. Marked aortic calcification in a young woman. *Am J Roentgenol* 73 52 1955
- 1239 RABINOWITZ, J. Stick and lip. *Scient. Am* 194 (No 5) 109 May 1956
- 1240 RAGAN, C. AND BORDLEY, J. III. Accuracy of

- in direct phonocardiography. *Am Heart J* 16: 930, 1938
- 1278 ROLLETON H D Heart showing a muscular band passing between the two ventricular papillares of the left ventricle and capable of acting as a moderator) and J Anat and Physiol 32: 91, 1936
- 1279 ROLLETON H D a The history of mitral stenosis. *Brit Heart J* 3: 1, 1941 b *Cardiac Diseases since Harvey's Discovery*. University Press, Cambridge, 1939
- 1280 ROSEN C A note on the reception of the stethoscope in England. *Bull Hist Med* 7: 93, 1939
- 1281 ROSENTHAL J and FRIEDMAN I Pathology of the mitral valve in the older age groups. *Am Heart J* 31: 346, 1946
- 1282 ROSENTHAL J and KILICKI I Aortic arch aneurysms: diminution or absence of pulsations arising from the arch of the aorta. *Arch Int Med* 62: 61, 1933
- 1283 ROSENTHAL J and KILICKI I and HARVEY J C The problem of fever in patients with valvular heart diseases. *J Am Med* 128: 1, 1936
- 1284 ROUSSEAU J Annot. des lésions du cœur. Paris: 1800, 1802
- 1285 ROUSSEAU J M Du claquement d'ouverture de la mitrale. *étude clinique, étiologique et pathogénique*. Paris: 1860
- 1286 ROUSSEAU D Remarque sur les signes d'insuffisance du cœur. *Arch Mal Cœur* 1: 44, 1933
- 1287 ROUSSEAU D MAMOT H and LEMAITRE J Rôle complet et auriculaire. *Butter* (à propos de deux observations personnelles). *Arch Mal Cœur* 1: 399, June 1936
- 1288 ROWE P D LEAD I and HEINTZ J D Atrial tetralogy of Fallot, a non-cyanotic form with increased lung vascularity. *Circulation* 39: 230, 1939
- 1289 RUDOLPH A M and MOYER F E Latent ductus arteriosus in the first year of life: clinical and hemodynamic observation in 2 patients. *Am Heart J* 16: 979, 1939
- 1290 RUDOLPH A M, MOYER F E and COOPER W T Intracardiac left to right shunt with pulmonary stenosis. *Am Heart J* 48: 505, 1934
- 1291 RUKAVINA J F, BLANK H D, JAKES C E, FALLS H F, CREECH J H and CLEGG A C Immune system: unambiguous: a review and an experimental genetic and clinical study of organ with particular emphasis on the familial form. *Medicine* 35: 233, 1936
- 1292 RUMER R F Anatomy and physiology of ventricular function. *Physiol Rev* 26: 400, 1936
- 1293 RUMER R F *Causes of the heart disease*. Philadelphia: 1930
- 1294 RUMER R F Symposium on cardiovascular and circulation. 1939
- 1295 RUMER R F, BANK P S and CLARK R M Direct writing heart phonorecorder. *Am J Physiol* 73: 1932
- 1296 RUMER R F, ENLAW H I and NASH A A Movement of the mitral valve. *Circulation* 16: 4, 1936
- 1297 RUMER R F, ENLAW H I and NASH A A Shrinkage of the heart in anesthetic thoracotomized dogs. *Circulation* 16: 2, 1936
- 1298 RUMER R F, SPARKMAN D R, KELLEY I E, BRYAN I I, BRICE R R, WILSON G B and BRIDGES W C Variability in detection and interpretation of heart murmurs: A comparison of auscultation and phonocardiography. *Am J Physiol* 74: 19, 1937
- 1299 RUMER R F, TIDWELL R A and KELLEY I E Sonographic recording of murmurs during acute myocarditis. *Am Heart J* 48: 330, 1934
- 1300 RUMER R F, KELLEY I E and TIDWELL R A Charles C Thomas Springfield, 1936
- 1301 RUMER R F, SCHIFFLEY C H and LEWIS J J Studies of the mitral valve I Anatomic feature of the normal mitral valve and its associated structures. *Circulation* 6: 30, 1936
- 1302 RUMER R F, SCHIFFLEY C H and LEWIS J J Studies of the mitral valve II Certain anatomic features of the mitral valve and its associated structures in mitral stenosis. *Circulation* 14: 379, 1936
- 1303 RUMER R F, SCHIFFLEY C H, LEWIS J J and KILICKI I W Guidelines to the committee in operations upon the mitral valve. *New Staff Meet. Mass. Clin.* 39: 99, 1941
- 1304 RUMER R F An analysis of the variable systolic time of the first heart sound in the mitral heart block. *Ann Int Med* 6: 187, 1936
- 1305 RUMER R F An auricular diastolic murmur with heart block in old patients. *Am Heart J* 32: 55, 1946
- 1306 RUMER R F The variable location of the first heart sound in auricular fibrillation. *Am Heart J* 31: 157, 1936
- 1307 RUMER R F and LEWIS J J Clinical aspects of calcification of the mitral annulus fibrous. *Arch Int Med* 78: 311, 1936
- 1308 RUMER R F Comparative morphological and pathological examination of the mitral valve in humans. *Circulation* 17: 465, 1936
- 1309 RUMER R F and KILICKI I The perforation of the interventricular septum following acute myocardial infarction: a report of four cases diagnosed ante mortem. *Ann Int Med* 11: 60, 1936
- 1310 RUMER R F, LEHRER H F and LEHRER H F Lehrbuch der Pathologie des Herzes. Leipzig: 1939
- 1311 RUMER R F, LEHRER H F and LEHRER H F The heart as a lower chamber. 1

- H AND JANCMANN A G Congenital malformations of the cardiovascular system in a series of 6053 infants *Pediatrics* 15 12 1955
- 1271 RICHARDS M R MERRITT K K SAMUELS M H AND JANCMANN A G Frequency and significance of cardiac murmurs in the first year of life *Pediatrics* 15 169 1955
- 1272 RICHARDSON B W Intrathoracic intubation a new departure in physical diagnosis *Lancet* 2 1037 1892
- 1273 RICHARDSON B W *Med Times and Gaz* 2 440 1869
- 1274 RICHIERAND A B *Anciens des Pancloukes Dictionnaire des Sciences Medicales* 1812
- 1275 RIST I The acoustic analysis of percussion sounds *Ann med* 21 19 1927
- 1276 RIVERO CARVALLO J M El diagnostico de la estenosis tricuspidea *Arch Int cardiol Méx* 20 1 1950
- 1277 RIVERO CARVALLO J M Semiología de las lesiones tricuspideas *Arch Int cardiol Méx* 21 567 1951
- 1278 RIVERO CARVALLO J M Signos para el diagnostico de las insuficiencias tricuspideas *Arch Inst cardiol Méx* 16 531 1946
- 1279 RIVERO CARVALLO J M CARRAL R AND JAIME M H R I tenosis relativa de la tricuspidea *Arch Int cardiol Méx* 21 47 1951
- 1280 ROBB C P *Atlas of Angiocardiography* Prepared for American Registry of Pathology Armed Forces Institute of Pathology Washington D C pp 46-57 1951
- 1281 ROBB J S AND ROBB R C The normal heart Anatomy and physiology of the structural units *Am Heart J* 23 455 1942
- 1282 ROBERTS J T Diagnosis of congenital heart disease before birth and hereditary factor in congenital heart disease *J Tech Method* 15 101 1938
- 1283 ROBERTS J T Dynamic and circulation of heart muscle cardiac reserve and the cardiac cycle In Sodeman W A *Pathologic Physiology* p 45 W B Saunders Co Philadelphia 1951
- 1284 ROBERTS J T *Medioneurosis aortae idiopathica cystica* Report of a case with healed dissecting aneurysm *Am Heart J* 18 154 1939
- 1285 ROBERTS I N SMILEY J R AND MANNING G W A comparison of direct and indirect blood pressure determination *Circulation* 8 232 1953
- 1286 ROBERTS S R A study of arterial sound *J A M A* 873 1917
- 1287 ROBINOW M AND HARPER H T Jr Functional mitral stenosis *Ann Int Med* 17 523 1942
- 1288 ROBBARD S An artificial circulation model demonstrating cardiovascular dynamic *J Lab & Clin Med* 38 267 1951
- 1289 ROBBARD S Flow through collapsible tubes augmented flow produced by resistance at the outlet *Circulation* 11 290 1955
- 1290 ROBBARD S Hydrodynamics illustrated in an artificial circulation model variable aneurysms coarctation sphygmomanometry coronary flow *J Appl Physiol* 5 191 1952
- 1291 ROBBARD S Mechanism of the systolic murmur and the aortic stenosis of aortic stenosis and coarctation *Am J Physiol* 5 191 1955
- 1292 ROBBARD S The significance of the intermediate Korotkoff sounds *Circulation* 8 600 1953
- 1293 ROBBARD S Symposium on cardiovascular sound *Circulation* 16 282 1957
- 1294 ROBBARD S AND CIESELSKI J Mechanism of auscultatory gap or doubling of arterial sound in aortic stenosis *Abstract Circulation* 10 930 1957
- 1295 ROBBARD S AND MARGOLIS J The auscultatory gap in arterio sclerotic heart disease *Circulation* 15 550 1957
- 1296 ROBBARD S AND MARCOIS J The significance of the intensity and time of appearance of the Korotkoff sound in aortic fibrillation *Circulation* 11 510 1956
- 1297 ROBBARD S MENDELSON C F AND ELIABRE E I Vibration analysis of heart sound and murmurs *Cardiologia* 27 144 1955
- 1298 ROBBARD S RUBINSTEIN H M AND ROENBLUM S Arrival time and calibrated contour of the pulse wave determined indirectly from recording of arterial compression sound *Am Heart J* 63 205 1957
- 1299 ROBBARD S AND SAKI H Flow through collapsible tubes *Am Heart J* 40 715 1953
- 1300 ROBBARD S AND SHAEFER A B Muscular contraction in the infundibular region as a mechanism of pulmonary stenosis in man *Am Heart J* 51 88, 1956
- 1301 ROBBARD S AND WILLIAMS E The dynamic of mitral insufficiency *Am Heart J* 48 521 1954
- 1302 ROBBARD S JAAKS R AND COOK W A study of hydrodynamics in simulated patent ductus arteriosus *Circulation Res* 3 613 1955
- 1303 RODRIGO F A Estimation of valve area and valvular resistance *Am Heart J* 45 1 1953
- 1304 ROGER H I Recherches cliniques sur la communication congénitale des deux coeurs par occlusion du septum interventriculaire *Bull Acad méd Paris* 8 1074 1879
- 1305 ROGERS H M EVANS I C AND DOMFIER I H Congenital aneurysm of the membranous portion of the ventricular septum report of two cases *Am Heart J* 40 781 1952
- 1306 ROGERS H M WALDRON B R MURPHY D F H AND EDWARDS J J Supraventricular stenosing ring of left atrium in association with endocardial sclerosis (endocardial fibroelastosis) and mitral insufficiency *Am Heart J* 40 774 1955
- 1307 ROGERS W M SIMMONS I BRONSLAY S B AND DETTERLING R A Jr Pulmonary valve

- in direct phonocardiography. *Arch Circulat*  
tion 16 930 1937
- 129 POLLESTON H D Heart showing a muscular  
band passing between the two ventricular apices  
of the left ventricle and capsule of acting  
as a moderator) and J Anat and Physiol 32  
1 149
- 130 POLLESTON H D The histology of mitral stenosis  
Brit Heart J 3 1 1931 & Cardiovascular  
Diseases since Harvey's Discovery University  
Press Cambridge 1934
- 131 POSEY C A note on the reception of the 10th  
copen in England Bull Hist Med 7 93 1939
- 132 ROBERTS J AND FRICKE I Pathology of the  
mitral valve in the different age groups. *Am Heart*  
J 31 316 1941
- 133 POSEY R B AND MICHENER A Aortic arch  
valvulosis diminishes or abolishes the valvular  
lesions arising from the arch of the aorta. *Arch*  
Int Med 92 51 1933
- 134 POSEY R B AND MICHENER A AND HARVEY J C  
The problem of fever in patient with valvular  
disease. *Am Heart J* 18 1 1935
- 135 POINCARÉ I Analyse des bruits du coeur Paris  
thesis No 20 1937
- 136 POINCARÉ R J M Du claquement d'ouverture de  
la mitrale étude clinique (médicologie et patho-  
logique) Paris the 14 1938
- 137 POINCARÉ I Remarques sur le syndrome du cœur  
du lac la pericardie du canal artériel. *Arch*  
mal coeur 30 384 1933
- 138 ROBERT D MONT H AND LEMAY J Bloc  
complet et auriculaire flutter à propos de deux  
observations personnelles. *Arch mal coeur* p  
379 June 1936
- 139 ROSS R D AND LADD J D AND HEATH J D The  
cardiac tetralogy of Fallot an anatomic form with  
increased lung vascularity. *Circulation* 12 230  
1930
- 140 FRIEDMAN A M AND MAYER E F Laboratory studies  
in atrial flutter in the first year of life chemical and  
hemodynamic observation in 71 patients. *Arch*  
Circulat (Circulation) 16 337 1937
- 141 RUSSELL A M AND BARNES J A AND DEER  
H T Intracardiac left to right shunt with pul-  
monary stenosis. *Am Heart J* 48 508 1933
- 142 FUKUDA S C BLOCK W D AND JONES C J  
PALLS H F CAREY J H AND CARTER A C  
Primary systemic amyloidosis a review and an  
experimental genetic and clinical study of 29  
cases with particular emphasis on the familial  
form. *Medicine* 30 229 1936
- 143 FRIEDMAN E F Anatomy and physiology of  
ventricular function. *Physiol Rev* 36 400 1936
- 144 FRIEDMAN E F Cardiac Diagnostics (Physiologic  
Apparatus) W B Saunders Co Philadelphia 1935
- 145 FRIEDMAN E F Experimental catheterization  
in the circulation. *Am Heart J* 31 270 1941
- 146 FRIEDMAN E F AND FRIEDMAN R M  
Direct writing heart sound recorder. *Am J Physiol* 23 33 1937
- 147 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A Measurement of the mitral valve. *Circulation*  
16 437 1935
- 148 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A Shrinkage of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 149 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 150 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 151 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 152 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 153 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 154 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 155 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 156 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 157 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 158 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 159 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 160 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 161 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 162 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 163 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 164 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 165 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 166 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 167 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 168 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 169 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 170 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 171 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 172 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 173 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 174 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 175 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 176 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 177 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 178 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 179 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 180 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 181 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 182 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 183 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 184 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 185 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 186 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 187 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 188 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 189 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 190 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. *Circulation Res* 2 155  
1934
- 191 FRIEDMAN E F AND FRIEDMAN R M AND NASH  
A A A Study of the heart in anesthetized  
thoracotomized dogs. <

- Contribution to Cardiodynamics* Henry Frowde and Hodder & Stoughton London 1922
- 1341 SAMPMSON J J McCALLA R L KERR W J Phonocardiography of the human fetus *Am Heart J* 1 717 1926
- 1341A SANDERS R J NEUBERGER K T AND RAVIN A Rupture of papillary muscle occurrence of rupture of posterior muscle in posterior myocardial infarction *Dis Chest* 31 316 1957
- 1342 SANGHVI L M SHARMA R AND MISRA S N Cardiovascular disturbances in chronic severe anemia *Circulation* 15 373 1957
- 1343 SANSON A L *Physical Diagnosis of the Heart* Philadelphia P Blakiston 1891
- 1344 SANSON A L *Valvular Disease of the Heart* pp 62 and 117 J & A Churchill London 1886
- 1345 SANSON A L On the cause and significance of reduplication of the sounds of the heart *Med Times Gaz* 2 58 1891
- 1346 SARNOFF S J Personal communication
- 1347 SARNOFF S J Second symposium on cardiovascular sound *Circulation in press*
- 1348 SARNOFF S J CASE R B WELCH C H JR BRAUNWALD L AND STAINSBY W N Observations on the performance characteristics and oxygen debt in a non fasting metabolically supported isolated heart preparation *Am J Physiol* in press
- 1349 SATO A AND MORIWAKI Y Difference of intensity of heart sounds expre ed numerically *Tohoku J Exper Med* 38 53 1940
- 1350 SATO A MORIWAKI Y AND MINAKAWA Quantitative estimation of normal heart sounds in healthy children *Tohoku J Exper Med* 38 55 1940
- 1351 SAYAT F Note sur la communication des mouvements vibratoires par les liquides *Ann de chimie et de physique* 31 283 1826
- 1352 SAYAT F On some acoustic phenomena produced by the motion of liquids through short efflux tube *Phil Mag* 7 186 1854
- 1352A SAWYER C C POOL R S BECK W C AND DANIEL L H JR Cor triatriatum *Am J Med* 23 798 1957
- 1353 SCADDING J G AND WOOD I Systolic clicks due to left sided pneumothorax *Lancet* 2 1208 1939
- 1354 SCHAEFER R D AND LITTLE R C The first heart sound in ventricular contraction arising from the apex and base *Proc Soc Exper Biol & Med* 85 639 1954
- 1355 SCHAEFER *Constat's Jahresbericht* Vol 2 p 100 1869
- 1356 SCHERF D AND BROOKS A M The murmurs of cardiac aneurysm *Am J M Sc* 218 389 1949
- 1357 SCHILDER D P AND HARVEY W P Confusion of tricuspid incompetence with mitral insufficiency—a pitfall in the selection of patients for mitral surgery *Am Heart J* 54 352 1957
- 1358 SCHILDER D P HARVEY W I AND HUFNAGEL C A Rheumatoid spondylitis and aortic insufficiency *New England J Med* 255 11 1956
- 1359 SCHILERO A J ANTZIS E AND DUNN J Friedreich's ataxia and its cardiac manifestations *Am Heart J* 44 905 1952
- 1360 SCHMIDT E Heart sounds audible at a distance as described in 1654 and one in 1951 *Acta med Scandinav* 266 (supp) 887 1952
- 1361 SCHMIDT VOIGT J Herzschalldiagnostik in Klinik und Praxis Georg Thieme Stuttgart 1961
- 1362 SCHWIMCKE A Endokardiale Thrombierung bei Aortenm. insuffizienz *Virchows Arch path Anat* 192 50 1905
- 1363 SCHWITZER M A AND BAYER C A Dissecting aneurysm of the aorta in young individuals particularly in association with pregnancy with report of a case *Ann Int Med* 20 486 1944
- 1364 SCHOELMERICH P AND CHERL H Herzschallstudien beim Targeweckel *Zt chr Krie kuf forsch* 40 211 1961
- 1365 SCHRATT W C JR AND LISA J R Duplication of the mitral valve Case report and review of the literature *Am Heart J* 39 136 1950
- 1366 SCHROEDER H *Hyperlensic Diseases* Lea & Febiger Philadelphia 1963
- 1367 SCHWABER H B J AND LUBIE P R Pulmonary valvotomy—description of a new operative approach with comments about the diagnostic characteristics of pulmonary valvular stenosis *J Thoracic Surg* 25 173 1953
- 1368 SCHWAR L SMILFY C I AND MEYER W I Aphythorax crunch in analysis of 106 cases in 324 Army sepiatees *Ann Int Med* 31 225 1949
- 1369 SCHWARTZ H AND CANELLI F R Spontaneous rupture of papillary muscle of the left ventricle *Am Heart J* 40 364 1960
- 1370 SCOTT J T Mediastinal emphysema and left pneumothorax *Dis Chest* 32 1957
- 1371 SCOTT J T AND MURPHY E A Mammary souffle of pregnancy *Abstract Circulation* 18 936 1967
- 1372 SEGAL J HARVEY W P AND HUFNAGEL C A clinical study of one hundred cases of severe aortic insufficiency *Am J Med* 21 200 1956
- 1373 SEGALL H N On writing symbols to describe cardiovascular sounds and murmurs while listening *Am Heart J* 33 706 1947
- 1374 SERRES M LEQUIME J AND DENOLIN H L activation precoce de certains coeurs hyper excitables *Etude de l'onde S de l'électrocardiogramme* *Cardiologia* 8 113 1944
- 1375 SELLING T Untersuchungen des Perikardions schalles *Deutsches Arch klin Med* 90 163 1907
- 1376 SELZER A Defects of the cardiac septums *J A M A* 154 129 1954
- 1377 SELZER A WILLETT F M McCAUGHEY D J AND FEICHTWEIR T V Uses of cardiac catheter

- terization in acquired heart disease. *New Eng Land J Med* 257 11 1957
- 1328 SWALL H A common modification of the first sound of the normal heart imitating that heard with mitral stenosis. *Am J M S* 138 10 1909
- 1329 SHARPEY-SCHAEFER E I Effects of Valvulae manoeuvre on the normal and failing circulation. *Brit M J* 1 673 1933
- 1330 SHARPEY-SCHAEFER E I Letter. *Lancet* 1 237 1940
- 1331 SHEARN M A AND LINDHART B Disminished lupus erythematosus. *Anal* 1 of 31 cases. *Arch Int Med* 90 90 1930
- 1332 SHEARN M A TARR F AND RETAND D A The significance of changes in amplitude of the first heart sound in children with A V block. *Circulation* 7 839 1953
- 1333 SHELTON P B AND DICE J The development of the telescope. *Bull N Y Acad Med* 11 608 1935
- 1334 SHEPHERD J T WELDMAN W H BIRKE F C AND WOLF F H Hemodynamics in patent ductus arteriosus without a murmur. *Circulation* 11 404 1953
- 1335 SHIBLEY G S New auscultatory sign found in consolidation or collection of fluid in pulmonary disease. *Chinese M J* 36 1 1922
- 1336 SHIGERU SATOMURA Ultra sonic Doppler method for the inpection of cardiac function. *J Acou Soc Am* 33 1187 1957
- 1337 SHILLITO F H CHAMBERLAIN F I AND IRVY R I Cardiac infarction. *J A M A* 118 79 194
- 1338 SCHULER R H ENGER C LUNNIN R I MOSS W C AND JOHNSON V The differential effect of reparation on the left and right ventricles. *Am J Physiol* 137 670 1947
- 1339 SHIMALKER H B JR Hemangioma of the liver. *Surgery* 11 209 194
- 1340 SIECKE H Anspannungzeit Ausbreitungzeit und mechanische Stille bei komplettem AV Block. *Ztschr Kreislaufforsch* 44 731 1953
- 1341 SIECKE H Ueber den Einfluss der IQ Zeit auf die Amplitude der Herzton sowie andere beim kompletten AV Block. *Ztschr Kreislaufforsch* 44 109 1953
- 1342 SIECKE H AND BERKE H F Relation between amplitude of first heart sound and duration of I Q interval in dog with atrioventricular block. *Am J Physiol* 191 469 1957
- 1343 SIKKE H AND JENSEN H F Relation of the difference in pressure across the mitral valve to the amplitude of the first heart sound in dog with atrioventricular block. *Am J Physiol* 192 123 1958
- 1344 SILBER L N PREC O CRESMAN N AND KATZ L N Dynamic of isolated pulmonary stenosis. *Am J Med* 10 71 1951
- 1345 SIMON M A AND HILL S F Calcification of the mitral valve annulus and its relation to functional valvular disturbance. *Am Heart J* 48 49 1954
- 1346 SIMON M A AND HILL S F Regurgitation from injury to the valves during muscular effort. *Brit M J* 2 167 1954
- 1347 SIMON M A MURPHY C F AND NEWMAN J V Multiple congenital arteriovenous aneurysms in the pulmonary circulation. *Bull John Hopkins Hosp* 78 93 1915
- 1348 SKIFFER I AND FLINT F J Symmetrical arterial occlusion of upper extremities. Lead and neck as rare syndrome. *Brit M J* 2 9 1957
- 1349 SKODA J *Tracillat* and *Pericussion*. Translated by W O Markham from 4th German edition. London and Blackton. Philadelphia 1951
- 1350 SKODA J Ueber die Erscheinungen an denen sich die Verwachsung des Herzens mit dem Herbeutel an lebenden Menschen erkennen lässt. *Ztschr d k k Gesellchr d Verste Wien* 1 395 1852
- 1351 SLOAN A W CAMPBELL F W AND HENIFER A A Incidence of the physiological third heart sound. *Brit M J* 2 53 1957
- 1352 SLOAN A W AND CROFT J R Calibration of an electronic phonocardiograph. *Brit Heart J* 17 19 1957
- 1353 SLOAN A W AND WEHART M Canine extra sound in the dog. *J Physiol* 122 17 1953
- 1354 SLOAN A W AND WEHART M The effect on the human third heart sound of variation in the rate of filling of the heart. *Brit Heart J* 15 23 1955
- 1355 SLOAN R D AND CHERRY R N Congenital pulmonary arteriovenous aneurysm. *Am J Roentgenol* 70 183 1953
- 1356 SMITH A I Recording and reproduction of a fetal heart murmur confirmed after birth. *Arch Pediatr* 68 549 1951
- 1357 SMITH A I Stethogram and recorded die of fetal heart sounds in a twin pregnancy. *Am J Obst Gynec* 42 908 1951
- 1358 SMITH D F AND MATTHEW M B Aortic valvular stenosis with coarctation of the aorta with special reference to the development of aortic stenosis upon congenital bicuspid valves. *Brit Heart J* 17 195 1955
- 1359 SMITH L T A New form of stethoscope. *Brit M J* 1 99 1954
- 1360 SMITH C Latent ductus arteriosus with pulmonary hypertension and reversed ductus. *Brit Heart J* 15 233 1955
- 1361 SMITH H J FRY H F AND BAKER I J A study of the movements of heart valves and first heart sound. *Ann Int Med* 33 13 1950
- 1362 SMITH J B Observations on the mechanism of the physiological third heart sound. *Am Heart J* 28 661 1944

- 1409 SMITH J R EDWARDS J C AND KOUNTZ W B  
The use of the cathode ray for recording heart sounds and vibrations III Total cardiac vibrations in one hundred normal subjects *Am Heart J* 21 228 1941
- 1410 SMITH J R GILSON A S AND KOUNTZ W B  
The use of the cathode ray for recording heart sounds and vibrations II Studies on the muscular element of the first heart sound *Am Heart J* 21 17 1941
- 1411 SMITH J R AND KOUNTZ W B Total cardiac vibrations in aged hearts and in coronary disease *Proc Soc Exper Biol & Med* 47 303 1941
- 1412 SMITH J R KOUNTZ W B AND GILSON A S  
A consideration of extra valvular elements in the first heart sound *Proc Soc Exper Biol & Med* 43 256 1940
- 1413 SMITH S M. Pericardial knock *Brit M J* 1 78 1918
- 1414 SOLOFF L The heart in rheumatoid arthritis Paper presented at 9th International Congress on Rheumatic Diseases Montreal June 28 1957
- 1415 SOUS COHEN M The apical heave and its incidence in healthy inductees *Am J M Sc* 210 333 1945
- 1416 SOLOFF L A STALFNER H M AND ZATCHEV J  
Ebstains disease Description of the heart of the first case diagnosed during life *Am J M Sc* 233 23 1957
- 1417 SOLDER C R LEARSON C M AND ADAMS H D  
Aortic deformity simulating mediastinal tumor subclinical form of coarctation *Dis Chest* 20 35 1951
- 1418 SOULIÉ P BOUCHARD F CORNU C AND BRIAL E  
Occlusion du canal artériel par cathétérisme Intérêt pronostique *Bull et mém Soc méd hôp Paris* p 547 1947
- 1419 SOULIÉ P COUBET J AND BRIAL M Syndrome de Wolff Parkinson et White et dérivation précoarctale *Arch mal coeur* 40 455 1947
- 1420 SOULIÉ P DI MATTEA P AND CHICHE I  
Traumatismes précordiaux non pénétrants La rupture septale L'anévrysme apical *Arch mal coeur* 39 41 1946
- 1421 SOULIÉ P LAURENS P BOUCHARD F CORNU C AND BRIAL E Enregistrement de pressions et des bruits intracardiques à l'aide d'un micromanomètre *Bull et mém Soc méd hôp Paris* p 713 1957
- 1422 SOULIÉ P ROULIER D AND BERNAL P Communication interventriculaire avec insuffisance aortique (diagnostic différentiel de la persistance du canal artériel) *Arch mal coeur* 42 765 1949
- 1423 SOUTHWORTH J L McKEUSICK V A PEIRCE E C II AND RAWSON F L JR Ventricular fibrillation precipitated by cardiac catheterization complete recovery of the patient after 45 minutes *J A M A* 143 717 1950
- 1424 SPICKNALL C G AND BINFORD C H Healed dissecting aneurysm of the aorta with signs of aortic insufficiency A case report *Mil Surg* 102 47 1948
- 1425 SPIEGEL R J LONG J B AND DEXTER L  
Clinical observations in patients undergoing finger fracture mitral valvulotomy auscultatory changes electrocardiographic observation *Am J Med* 12 626 1952
- 1426 SPÖRR H Starke Dissoziation zwischen dem Ende der elektrischen und mechanischen Systolendauer bei Kängurus *Cardiologia* 11 2/8 1956
- 1427 SPRAGUE H B The art of auscultation of the heart *Practitioner* 176 241 1956
- 1428 SPRAGUE H B A new combined stethoscope chest piece *J A M A* 86 1900 1928
- 1429 SPRAGUE H B AND ONGLEY I A The clinical value of phonocardiography *Circulation* 9 127 1954
- 1430 SPRAGUE H B AND RAFFAPORT M B The effects of improper fitting of stethoscope to ears on auscultatory efficiency *Am Heart J* 43 713 1952
- 1431 STAHL R AND ENTZIAN W Klinisches und Experimentelles über das intra und extrakardiale Mithlengeräusch *Ztschr klin Med* 100 232 1924
- 1432 STALLWORTH J M MARTIN J H AND FOSTLE THWAIT R W Aspiration of the heart in air embolism *J A M A* 143 1250 1950
- 1433 STALFNER H AND WIEL C Observationum Rariorum Medic Anatomie Chirurgicarum Cent I obs 36 Quoted by Morgagni *loc cit* p 394
- 1434 STARZEL T E AND GAERTNER R A Chronic heart block in dog A method for producing experimental heart failure *Circulation* 12 259 1956
- 1435 STEAD C A AND KUNDEL P Factors influencing the aortic murmur and the intensity of the first heart sound *Am Heart J* 10 261 1939
- 1436 STEEL G The murmur of high pressure in the pulmonary artery *Med Chronicle* 0 182 1888
- 1437 STEINBERG I Aneurysm of aortic sinuses with pseudocoarctation of aorta *Brit Heart J* 18 55 1956
- 1438 STEINBERG I Diagnosis of congenital aneurysm of the ventricular septum during life *Brit Heart J* 19 8 1957
- 1439 STEINBERG I AND FINBY N Clinical manifestations of the unperforated aortic aneurysm *Circulation* 14 115 1956
- 1440 STEINFELD L F KIRCHNER I A MINOR J B KUHN I A AND GORDON A J Continuous pressure difference across stenotic mitral and aortic valves recorded during left heart catheterization Abstract *Circulation* 16 941 1957
- 1441 STRINZEC S M HANSKY S T ALMURUNG

- W M THOMAS T C Phonocardiographic changes in mitral stenosis before and after valvulotomy: a correlation with mitral valve size *Am Heart J* 52 730 1957
- 144 STEINBERG I A HERTZMAN M P AND HERRMAN C R Unusual mitral murmurs of anterior cup aortic regurgitation *Report of 10 cases* *Am Heart J* 51 163 1956
- 145 STEWART C F AND FERNER H B A note on pericardial involvement in coronary thrombosis *Am Heart J* 51 21 1958
- 146 STEWART H A Discussion of paper of J M Under *Bull Johns Hopkins Hosp* 1909
- 147 STEWART I M C Aortic murmurs in healthy young adults *Brit Heart J* 13 401 1951
- 148 STILL C F *Common Disorders and Diseases of Children* 2nd ed p 333 Frowde London 1909
- 149 STILL C F 3rd ed p 430 Oxford University Press 1915
- 150 STOKES W *Diseases of the Heart and the Aorta* 1st ed C Blackie London 1950
- 151 STRAUSS A AND MORGAN R Phonocardiographic registration of the aortic murmurs on peripheral arteries due to organic arteriopathies *Cardiolgia* 23 230 1952
- 152 STRASSER M (a) Cultures a source of sound in liquid *J Acoust Soc Am* 28 20 1956
- 153 STRUBER M MARTINEZ F AND KEILY J J Factors influencing the time of onset of the first heart sound in normal subjects *Am Heart J* 54 84 1957
- 154 STRONG J A Thyrotoxicosis with ophthalmoplegia myopathy Wolff Larkin and White and dome and pericardial friction *Lancet* 2 9 1949
- 155 STUCKEY D Innocent aortic murmurs of aortic origin *M J Australia* 44 58 1957
- 156 STUCKEY D Dwyer B and Walker H Cardiac murmurs in aortic aneurysm *M J Australia* 44 98 1957
- 157 SYDNER C C Aortography of the heart with calcium annulus fibrous (mitralis) *Acta Radiol* 18 27 1951
- 158 STRAWL B Symposium on cardiovascular sound *Circulation* 18 20 and 414 1957
- 159 SWANWICK B AND LEITCHMAN I The electrocardiographic pattern of hypoxaemia with and without hypocalcaemia *Circulation* 8 401 1953
- 160 SWANWICK C C LITTLE D M JR McCLELLAN J Measurement of mechanical and electrical event of the cardiac cycle I Effect of tachycardia pre-anesthetic medication and ethyl ether anesthesia *Am J M Sc* 232 619 1956
- 161 SYDER H W The decay of acoustical and the use of the bisaural stethoscope *Lancet* 1 383 1907
- 162 TALENT S A Pericardial communication
- 163 TALENT S A C MELL B F AND WALSH B J Phonocardiographic studies of early rheumatic mitral disease *Am Heart J* 20 25 1950
- 164 TALENT H B (a) *Genital Malformations of the Heart* Commonwealth Fund New York 1914
- 165 TALENT H B AND BING R J Complete transposition of the aorta and a less position of the pulmonary artery clinical physiological and pathological findings *Am Heart J* 37 401 1913
- 166 TALENT H B AND GRUBBS J H Severe aortic insufficiency in a situation with a congenital malformation of the heart of the Littenberger type *Bull Johns Hopkins Hosp* 65 14 1910
- 167 TAYLOR W C The incidence and significance of aortic cardiac murmurs in infants *Arch Dis Childhood* 25 1 1953
- 168 TAYLOR D (a) Symmetrical hypertrophy of the heart in young adults *Brit Heart J* 20 1 1958
- 169 TAYLOR W C The early diastolic heart sound (the so-called third heart sound) *Tr Am Physician* 11 34 1918
- 170 TAYLOR W C Further observations on the third heart sound *Arch Int Med* 4 27 1909
- 171 TAYLOR W C Of the presence of a venous hum in the epigastrium in cirrhosis of the liver *Am J M Sc* 141 317 1911
- 172 TAYLOR W C On the early diastolic heart sound (the so-called third heart sound) *Briton Med & Surg J* 158 13 1908
- 173 TAYLOR W C Quelques remarques sur le troisième bruit du coeur *Arch mal coeur* 3 15 1910
- 174 TAYLOR W C Reflections on the interjection of aortic cardiac murmurs *Am J M Sc* 169 313 1911
- 175 TAYLOR W C Studies in (a) febrile (infective) endocarditis *Johns Hopkins Hosp Reports* 22 1 1906
- 176 TAYLOR W C AND MCCLELLAN W C Experimental studies of cardiac murmurs *Am J M Sc* 133 493 1907
- 177 THOMPSON W I AND FRANK S A Systolic gallop rhythm clinical study *New England J Med* 213 1071 1955
- 178 THOMPSON J J CARRALL R DE VINCENZI AND FERRAZZI D The clinical diagnosis of the aortic valve a study of eighteen cases *Am Heart J* 47 601 1954
- 179 THOMPSON J J *Leber den Depressen in der Cruralis* In *Effizienz der kontraktiven Herz klin* *Wochenschr* 9 3 1955
- 180 THOMPSON J A Case of aneurysm in the aorta in the orbit cured by the ligature of the common carotid artery *Med Chir Tr Roy Med & Chir Soc* 2 1 1913
- 181 THOMPSON J W *Deux micrologie du coeur et les cas de la* 13a *Mus on Paris* 1907 Cite 1 by Cato (231)
- 182 THOMPSON J W (a) Observations on the causes of the



- 1409 SMITH J R EDWARDS J C AND KOUNTZ W B  
The use of the cathode ray for recording heart sound and vibration III Total cardiac vibrations in one hundred normal subjects *Am Heart J* 21 228 1941
- 1410 SMITH J R GILSON A S AND KOUNTZ W B  
The use of the cathode ray for recording heart sounds and vibrations II Studies on the muscular element of the first heart sound *Am Heart J* 21 17 1941
- 1411 SMITH J R AND KOUNTZ W B Total cardiac vibrations in aged hearts and in coronary disease *Proc Soc Exper Biol & Med* 47 353 1941
- 1412 SMITH J R KOUNTZ W B AND GILSON A S  
A consideration of extra valvular elements in the first heart sound *Proc Soc Exper Biol & Med* 43 256 1940
- 1413 SMITH S M Pericardial knock *Brit M J* 1 78 1918
- 1414 SOKOLOFF L The heart in rheumatoid arthritis Paper presented at 9th International Congress on Rheumatic Disease Montreal June 28 1957
- 1415 SOLIS COREA M The viphernal crunch and its incidence in healthy inductees *Am J M Sc* 210 373 1945
- 1416 SOLOFF I A STAEFFER H M AND ZATCHEM J E  
Hearts disease Description of the heart of the first case diagnosed during life *Am J M Sc* 233 23 1957
- 1417 SOLLERS C R PEARSON C M AND ADAMS H D  
Aortic deformity simulating mediastinal tumor umbilical form of coarctation *Dis Chest* 20 35 1951
- 1418 SOULIÉ P BOICHARD F CORNI C AND BRIAL I  
Occlusion du cœnal artériel par cathétérisme Intervent préopératoire *Bull et mém Soc méd hôp Paris* p 547 1947
- 1419 SOULIÉ P COCHRAN J AND BRIAL M  
Syndrome de Wolff Parkinson et White et dérivation précoarctale *Arch mal coeur* 40 453 1947
- 1420 SOULIÉ P DI MATTEA P AND CHICHE P  
Traumatismes précoarctaux non pénétrants La rupture septale L'anévrisme apexien *Arch mal coeur* 39 41 1946
- 1421 SOULIÉ P LAURENS P BOICHARD F CORNI C AND BRIAL E  
Enregistrement des pressions et de bruits intracardiaques à l'aide d'un micromanomètre *Bull et mém Soc méd hôp Paris* p 713 1957
- 1422 SOULIÉ P ROLLIER D AND BERNAL P  
Communication interventriculaire avec insuffisance aortique (diagnostic différentiel de la persistance du cœnal artériel) *Arch mal coeur* 33 765 1949
- 1423 SOUTHWORTH J L MCKUSICK V A PEIRCE E C II AND RAWSON F I JR  
Ventricular fibrillation precipitated by cardiac catheterization complete recovery of the patient after 45 minutes *J A M A* 143 717 1950
- 1424 SPICKALL C C AND BINFORD C H  
Healed dissecting aneurysm of the aorta with sign of aortic insufficiency A case report *Mil Surg* 102 47 1948
- 1425 SPIEL R J LONG J B AND DEXTER L  
Clinical observations in patients undergoing finger fracture mitral valvulotomy auscultatory changes electrocardiographic observations *Am J Med* 12 626 1952
- 1426 SPRORR H  
Stärke Dissoziation zwischen dem Ende der elektrischen und mechanischen Systole bei Kanarienvögeln *Cardiologia* 25 2 S 1956
- 1427 SPRAGLE H B  
The art of auscultation of the heart *Practitioner* 176 241 1956
- 1428 SPRAGLE H B  
A new combined stethoscope chest piece *J A M A* 86 1909 1926
- 1429 SPRAGUE H B AND OXLEY P A  
The clinical value of phonocardiography *Circulation* 9 127 1954
- 1430 SPRAGUE H B AND RAPPAPORT M B  
The effects of improper fitting of stethoscope to ears on auscultatory efficiency *Am Heart J* 43 713 1952
- 1431 STALL R AND ENTENH W  
Klinisches und Experimentelles über das intra und extrakardiale Mischgeräusch *Ztschr klin Med* 100 212 1924
- 1432 STALLWORTH J M MARTIN J B AND FOSTER THOMAS R W  
Aspiration of the heart in air embolism *J A M A* 143 1250 1950
- 1433 STALPERT VON DER WIEL C  
Observationum Rithorim Medice Anatomic Chirurgiarum Cent 1 ob 36 Quoted by Morgagni loc cit p 394
- 1434 STARZEL T F AND GAERTNER R A  
Chronic heart block in dog A method for producing experimental heart failure *Circulation* 12 259 1955
- 1435 STEAD F A AND HUKAEL F  
Factors influencing the aortic murmur and the intensity of the first heart sound *Am Heart J* 18 261 1939
- 1436 STELLA C  
The murmur of high pressure in the pulmonary artery *Med Chronicle* 9 152 1885
- 1437 STEINBERG I  
Aneurysm of aortic sinus with pseudocoarctation of aorta *Brit Heart J* 18 55 1956
- 1438 STEINBERG I  
Diagnosis of congenital aneurysm of the ventricular septum during life *Brit Heart J* 19 8 1957
- 1439 STEINBERG I AND FINBY N  
Clinical manifestation of the unperforated aortic aneurysm *Circulation* 14 115 1956
- 1440 STEINFELD I L KIRCHNER P A MINOR J B KUCH I A AND ORDON A J  
Continuous pressure difference across mitral and aortic valves recorded during left heart catheterization *Abstract Circulation* 16 931 1957
- 1441 STEINZEIG S M LINSAY S T ALTMURUNG

- 1513 WARREN J S Editorial Gallop rhythm Circulation 15 371 1957
- 1514 WARREN J S LEONARD J J and WEISLER A M Gallop rhythm Ann Int Med 48 540 1958
- 1515 WATSON T Lectures on the Principles and Practice of Physics p 611 Lea and Blanchard Philadelphia 1944
- 1516 WATSON W L Pulmonary arteriovenous fistula Surgery 9 19 194
- 1517 WEBB C A and McINTOSH J A Analysis of heart sound Proc of 1957 National Conference on Instrumental Methods of Analysis Instrument Society of America Pittsburgh Pa
- 1518 WEBER A Herzchallregistrierung Theodor Steinkopff Dresden and Leipzig 1944
- 1519 WEBER F I Can the clinical manifestations of congenital heart disease be interpreted with the general growth and development of the patient? Brit J Child Dis 35 315 1918
- 1520 WEBER T Physiologische und physiologische Experimente über die Entstehung der Geräusche in den Blutgefäßen Arch f physiol Heilk 14 26 1880
- 1521 WEINSTEIN B C and RHOODE L H Differential diagnosis of rheumatic fever in office practice J A M A 157 943 1946
- 1522 WEINSTEIN M S and LARSEN J F Mitral stenosis and its differential diagnosis by cardiac myxoma Am Heart J 38 61 1941
- 1523 WEINSTEIN W and LEE M Aortic diastolic murmurs without mitral stenosis Am Heart J 29 500 194
- 1524 WEISBERG A S and CATHART B M Pulmonary arteriovenous fistula and its long-term Ann Int Med 41 985 1954
- 1525 WEISBERG A S and JACOBSON C Regeneration von Herzmuskel und Herzkranzgefäßen mittel des Phonokopfs und ihre Beziehungen zum Ektrokardiogramm Z klin Med 72 910 1911
- 1526 WEISBERG D The mechanism and significance of the aortic and mitral regurgitation Circulation 15 27 0 1950
- 1527 WEISBERG C H JR BRIDGES J F and SWEET R J Hemodynamic effects of quantitative increased experimental aortic regurgitation Circulation 15 516 1950
- 1528 WELCH J J and KUNTER T D Effect of pressure fluctuation and of intervascular and interventricular septal defects on development of pulmonary vascular lesions Am J Path 24 79 1949
- 1529 WELLS B The mechanism of mitral stenosis by phonocardiography Brit Heart J 18 761 1954
- 1530 WELLS B The graphic configuration of innocent diastolic murmurs Brit J 15 23 1953
- 1531 WELLS B G Prediction of mitral pressure gradient from heart sound Brit J 15 31 1953
- 1532 WELLS B C and HART R I Congenital aortic stenosis: a study of the internal mammary artery Brit Heart J 19 135 1959
- 1533 WELLS B C PARRINGTON M B and STRICKER H B Graphic registration of aortic diastolic murmurs Am Heart J 37 28 1949
- 1534 WELLS B C PARRINGTON M B and STRICKER H B The sound and murmur in correlation of the aorta Am Heart J 38 67 1949
- 1535 WESTERMARK A Study of cardiac movements simultaneously recorded by roentgen cinematography and electrocardiography Acta radiol 22 373 1911
- 1536 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1537 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1538 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1539 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1540 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1541 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1542 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1543 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1544 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1545 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1546 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1547 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1548 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1549 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1550 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1551 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1552 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1553 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1554 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1555 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1556 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1557 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1558 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1559 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1560 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1561 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1562 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1563 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1564 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1565 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1566 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1567 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1568 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1569 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1570 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1571 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1572 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1573 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1574 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1575 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1576 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1577 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1578 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1579 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1580 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1581 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1582 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1583 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1584 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1585 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1586 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1587 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1588 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1589 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1590 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1591 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1592 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1593 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1594 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1595 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1596 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1597 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1598 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1599 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931
- 1600 WETTER F C and JACOBSON M Physiological aortic tricuspidal fusion Proc Physiol Soc 1931

- sounds produced by the action of the heart  
Trans Med Char Soc Edinburgh 3 205 1829
- 1478 TURRETTINI G De la propagation insolite et  
lointaine du souffle de l'insuffisance mitrale  
Arch mal coeur 11 489 1922
- 1479 TYLER D E The relationship between the  
pressure sustained by the various cardiac  
valves and the relative frequency of their  
involvement in rheumatic fever Am Heart J  
51 415 1956
- 1480 UNLEY M H Lutembacher's syndrome and a  
new concept of the dynamics of interatrial  
septal defect Am Heart J 24 315 1942
- 1481 ULRICH H L Report of a case of patent ductus  
arteriosus with some unusual features Acta  
med Scandinav suppl 196 160 1947
- 1482 URICCHIO J GOLDBERG H AND LIKOFF W  
Tricuspid regurgitation masquerading as mitral  
regurgitation in patients with pure mitral  
stenosis Abstract Circulation 11 945 1957
- 1483 VAMIL R J Ventricular aneurysms of the heart  
Preliminary report on some new clinical signs  
Am Heart J 49 934 1955
- 1484 VAIANALVA A M Opera 2 Vol Venice 1740
- 1485 VAN BOCAERT A AND VAN BOCAERT I A propos  
des alterations de l'électro cardiogramme dans  
la maladie de Friedrich Arch mal coeur 29  
630 1936
- 1486 VERNANT P NOVAHIE J SCHWEINERTH O  
JABESSE J BOLLHARD F MATHEY J BINET  
J P AND OLSTRIFFS G O Arch mal coeur  
48 277 1955
- 1487 VESALIUS A De corporis humani fabrica libri  
septem VI viii p 592 1543
- 1488 VIEU I Le souffle présystolique dans la sténose  
mitrale avec arythmie complete Arch mal  
coeur 40 442 1949
- 1489 VIEU I AND ILAT I Sur les circonstances  
d'apparition de l'éclat du premier bruit au cours  
de la diastole A V incomplete chez les  
mitraux Arch mal coeur 40 446 1947
- 1490 VIERORDT H Die angeborenen Herzkrankheiten  
Vienna 1898
- 1491 VIERORDT H Die Messung der Intensität der  
Herztöne Tübingen 1885
- 1492 VIERORDT H Kurzer Abriss der Perkussion und  
Auskultation Urban & Schwarzenberg, Berlin  
1930 1<sup>ed</sup> 1 1884
- 1493 VIERORDT H Medizinische aus der Geschichte  
Tübingen 1910
- 1494 VIETS H H In Webb Hymaker The Founders  
of Neurology Charles C Thomas Springfield  
1953
- 1495 VOGELFOEL L AND SCHRIRE V The role of aus-  
cultation in the differentiation of Fallot's  
tetralogy from severe pulmonary stenosis with  
intact ventricular septum and right to left  
interatrial shunt Circulation 11 714 1955
- 1496 VOGELFOEL L SCHRIRE V NELLEN M AND  
GOETZ R H The differentiation of the tet-  
ralogy of Fallot from severe pulmonary stenosis  
with intact ventricular septum and right to  
left shunt Angiology 8 215 1957
- 1497 VON GIERKE H E OESTREICHER H L FRANKE  
I K PARRACK H O AND VON WITTERN  
W W Physics of vibration in living tissues  
J Appl Physiol 4 686 1952
- 1498 WADE G ELIASCH H AND WERKO I On the  
Austin Flint murmur Acta med Scandinav  
142 (supp 266) 925 1952
- 1499A WADIA N H AND MONKTON G Intracranial  
bruises in health and disease Brain 80 407 1957
- 1499 WAKAT C S AND EDWARDS J E Developmental  
and pathologic considerations in persistent  
common atrioventricular canal Proc Staff  
Meet Mayo Clinic 31 487 1956
- 1500 WALDMAN S Brooklyn N Y Personal com-  
munication
- 1501 WALFAR V F Clinical localization of intra-  
cranial aneurysms and vascular anomalies  
Neurology 6 79 1956
- 1502 WALFAR V F AND ALIFORE G F Cirotid  
cavernous fistula surgery 39 411 1956
- 1503 WALLACH J B LUKASH I AND ANCRIST A A  
Occlusive aortic thrombi Am J Med 15  
77 1953
- 1504 WALLACE J D BROWN J R JR LEWIS D H  
AND DEITZ C W Acoustic snapping within  
the heart J Acous Soc Am 29 9 1957
- 1505 WATKINS W H Textbook of Cardiology 1<sup>ed</sup> 3  
p 81 Blanchard and Lea Philadelphia 1952
- 1506 WATHER R J STARAFY C W B ZERVAKIOTIS  
I AND CIBBONS C A Coronary arteriovenous  
fistula Clinical and physiologic report on two  
patients with review of the literature Am J  
Med 20 213 1957
- 1507 WARBURG E A method of determination of the  
undamped natural frequency and the degree of  
damping in a vibrating system of one degree  
of freedom by means of a square wave impact  
Acta physiol Scandinav 12 344 1949
- 1508 WARBURG L Cardiac murmurs audible at a  
distance—mitral stenosis—thrombosis of the  
right ventricle—pulmonary infarction Acta  
med Scandinav 107 593 1941
- 1509 WARREN H I DEWALL R A COHEN M  
VARCO R AND ILLFELD C W A surgical  
pathologic classification for isolated ventricular  
septal defects and for the Fallot's tetralogy  
based on observation made on 120 patients  
during repair under direct vision J Thoracic  
Surg 31 21 1957
- 1510 WARNER H R AND TOWNSEND A F Back flow  
in the aorta of patients with aortic insuffi-  
ciency studied with an indicator technique Ab-  
stract Circulation 16 917 1957

- B T Epigastric thrill and murmur as a clue of cirrhosis of the liver with general vascular signs of arteriovenous fistula Proc Staff Meet Mayo Clin 33 1938
- 1203 WOLFE TON W H Croonian lecture (1907) Phil Trans Roy Soc London p 1 1910
- 1204 WOLMAN M Hypertrophy of the branches of the pulmonary arteries and its possible relation with the so called primary pulmonary arterio sclerosis in two infants with hypertrophy of the right heart Am J Med Sc 220 133 1950
- 1205 WOLTER H H BYER O and QUERRMAN I Zur Beurteilung des Schweregrades von Mitralstenosen Ztchr Kreislaufforsch 44 1-1950
- 1206 WOOD A Acoustics Interscience Publishers Inc New York 1941
- 1207 WOOD F C JOHN ON J SCHWABEL T C JA HUI P T AND FISHER H F The diastolic heart beat Tr A Am Physician 31 95 1951
- 1208 WOOD P An appreciation of mitral stenosis I Clinical features Brit Med J 3 1051 1954
- 1209 WOOD P Investigations and results I 1113 1954
- 1210 WOOD P Congenital heart disease A review of its clinical aspects in the light of experience gained by means of modern technique (3rd Cire lecture) Brit Med J 2 639 and 643 1950
- 1211 WOOD P H Diseases of the Heart and Circulation ed 2 J B Lippincott Co Philadelphia 1956
- 1212 WOOD I Pulmonary hypertension Proc Royal Soc Med 42 190 1949
- 1213 WOOD P Incubation of phthisis of the lung Brit Heart J 20 77 1958
- 1214 WOOD P MURPHY O AND WILSON I A O Ventricular septal defect with a note on aortic Fallot tetralogy Brit Heart J 18 307 1954
- 1215 WRIGHT J L BIRNELL H O WOOD I H HAYE F A JR AND CLARKE O T Hemodynamic and clinical appraisal of coarctation during and even years after resection and endarterectomy of the aorta Circulation 24 800 1956
- 1216 WRIGHT W C Lancet's Deane pamphlet Millman New York 1950
- 1217 WYCKOFF J AND BENSON J Observation on an apical diastolic murmur associated with valvular disease best illustrated of right ventricular hypertrophy Tr A Am Physicians 60 280 1935
- 1218 YAMAKAWA K SHIMOMOTO Y KITAMURA K NAGAI T YAMAMOTO T AND ONITA S Intracardiac phonocardiography Am Heart J 47 471 1954
- 1219 YAMAKAWA K SHIMOMOTO Y NAGAI T KITAMURA K ONITA S AND YAMAMOTO T An attempt on the intracardiac phonocardiography Tohoku J Exper Med 58 311 1953
- 1220 YATER W M The plexus of Chiari's network Am Heart J 11 47 1936
- 1221 YATER W M Variation and anomalies of the venous valves of the right atrium of the human heart Arch Path 7 315 1953
- 1222 YIP J Y HARRIS D I LOWERY F W JR AND RYAN R F AND MURPHY I B Clinical and hemodynamic studies of aortic stenosis Circulation 13 640 1956
- 1223 ZAHN F W (Der einige anatomische Kennzeichen der Herzklappen insuffizienz Verhandl d Kongress Inn Med 23 351 1909)
- 1224 ZIEGLER R F The electrocardiogram in congenital valvular disease In Cardiac arrhythmia a symposium edited by (ed) H FARMER W B Saunders Co Philadelphia 1955
- 1225 ZIEGLER R F The importance of patent ductus arteriosus in infants Am Heart J 43 57 1952
- 1226 ZILVA A AND CLARK C Evaluation of murmurs in early rheumatic heart disease Am J Med 17 600 1954
- 1227 ZIEGLER R F Personal communication
- 1228 ZIEGLER R F JR AND HAYE F F The straining force exerted as an aid in the anatomic localization of cardiovascular murmurs and sounds Circulation 2 523 1940
- 1229 ZICKLER R LEHMANITZ S BERNY H AND SCHMIDT R M Perforation of the interventricular septum Arch Int Med 89 830 1959

- Henry Jackson Memorial Lecture } Circulation  
# 321-348 1952
- 1518 WIGGERS C J Factors determining the relative intensities of the heart sound in different auscultatory areas Arch Int Med 24 471 1919
- 1549 WIGGERS C J AND DEAN A I Jr The nature and time relations of the fundamental heart sounds Am J Physiol 88 476 1917
- 1550 WIGGERS C J A physiological investigation of the treatment in hemoptysis Arch Int Med 8 17 1911
- 1551 WIGGERS C J *Physiology in Health and Disease* ed 5 Lea & Febiger Philadelphia 1949
- 1552 WIGGERS C J Some factors controlling the shape of the pressure curve in the right ventricle Am J Physiol 33 382 1914
- 1553 WIGGERS C J Studies on the consecutive phases of the cardiac cycle II The laws governing the relative durations of ventricular systole and diastole Am J Physiol 66 479 1921
- 1554 WIGGERS C J AND DEAN A I The nature and time relation of the fundamental heart sounds Am J Physiol 42 476 1917
- 1555 WIGGERS C J AND DEAN A I Jr Principles and practice of registering heart sounds by direct methods Am J Med Sc 153 666 1917
- 1556 WIGGERS C J AND DEAN A Jr The registration of heart sounds from the exposed heart and large vessels A demonstration Proc Soc Exper Biol & Med 14 12 1916
- 1557 WIGGERS C J AND FEIN H Cardiodynamics of mitral insufficiency Heart 9 149 1922
- 1558 WIGGERS C J AND HATT I N The contour of the ventricular volume curves under different conditions Am J Physiol 68 439 1922
- 1559 WIGIE F D Duplication of the mitral valve Brit Heart J 19 296 1957
- 1560 WILDER R J AND MOSKOWITZ H I Left heart angiography in the diagnosis of mitral or aortic insufficiency Abstract Circulation 16 952 1957
- 1561 WILLIAMS A H AND GROPPER A I Interrelationships of cardiac output, blood pressure and peripheral resistance during normal respiration in normotensive and hypertensive individuals Circulation 4 278 1951
- 1562 WILLIAMS C J B *Memoirs of Life and Work* Smith Elder & Co London 1884
- 1563 WILLIAMS C J B *The Pathology and Diagnosis of Diseases of the Chest with New Researches on the Sounds of the Heart* J Churchill London 1835
- 1564 WILLIAMS C B, LANCE R L AND HECHT H H Postvalvular stenosis of the pulmonary artery Circulation 16 195 1957
- 1565 WILLIAMS C T Laennec and the evolution of the stethoscope Brit M J 2 6 1907
- 1566 WILLIAMS H B AND DODGE H I Analysis of heart sound Arch Int Med 38 685 1926
- 1567 WILLIAMS F A AND DRY T J *A History of the Heart and Circulation* W B Saunders Philadelphia 1948
- 1568 WILLIAMS F A AND KEY T L *Cardiac Classics* C V Mosby St Louis, 1941
- 1569 WILSON A B K AND FOTHERGILL J A recording sphygmomanometer Lancet 2 1029 1956
- 1570 WILSON F U AND JAMIESON R A The musical murmur of aortic insufficiency Heart 7 71 1918-1920
- 1571 WILSON R A Case of Chiari's network associated with a murmur resembling the bruit de Roger J A M A 111 917 1938
- 1572 WINCHELL P AND BASHOUR F Ventricular septal defect with aortic incompetence simulating patent ductus arteriosus Am J Med 20 761 1956
- 1573 WINDHOLZ F AND GRAYSON C L Intrusion of aortic root into mitral orifice in hypertensive disease radiologic observations on living persons Am Heart J 34 180 1947
- 1574 WINSOR T AND BURCH C E The electrocardiogram and cardiac state in active sickle cell anemia Am Heart J 29 685 1945
- 1574A WINTER B Pulmonic stenosis produced by compression of heart by anterior mediastinal tumor Am Heart J 55 15 1958
- 1575 WOLPERTH C C Personal communication
- 1576 WOLPERTH C C AND MARCOLIS A Asynchronous contraction of the ventricles in the so called common type of bundle branch block its bearing on determination of the site of the significant lesion and on the mechanism of the split first and second heart sounds Am Heart J 10 425 1935
- 1577 WOLPERTH C C AND MARCOLIS A Tallopp rhythm and the physiological third heart sound characteristics of sounds classification comparative incidence of various types and differential diagnosis Am Heart J 8 441 1933
- 1578 WOLPERTH C C AND MARCOLIS A The influence of auricular contraction on the first heart sound and the radial pulse Arch Int Med 40 1015 1930
- 1579 WOLPERTH C C AND MARCOLIS A Systolic gallop rhythm Am Heart J 19 129 1940
- 1579A WOLPERTH C C AND MARCOLIS A Heart sounds Chapter 26 of *Diagnosis and Treatment of Cardiovascular Disease* (W D Stroud ed) Philadelphia F A Davis 1945
- 1580 WOLPERTH C C, WOOD F C AND MARCOLIS A An auriculo-systolic murmur in the tricuspid area during convalescence from acute coronary occlusion Am J Med Sc 186 496 1933
- 1581 WOLFF B P Spontaneous interstitial emphysema of the lungs report of an additional case Ann Int Med 12 1250 1940
- 1582 WOLLAEGER I E, KEITH A M AND HORTON









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